ORAL MUCOSAL SYMPTOMS, SIGNS AND LESIONS IN THE END STAGE RENAL DISEASE AND NON-END STAGE RENAL DISEASE IN DIABETES MELLITUS PATIENT

Dissertation submitted to

THE TAMILNADU Dr.M.G.R.MEDICAL UNIVERSITY

In partial fulfillment for the Degree of

MASTER OF DENTAL SURGERY

BRANCH IX

ORAL MEDICINE AND RADIOLOGY

MARCH 2012

CERTIFICATE

This is to certify that this dissertation titled "**Oral Mucosal Symptoms, Signs and Lesions in the End Stage Renal Disease and Non-End Stage Renal Disease in Diabetes Mellitus Patient**" is a bonafide record of work done by **Dr.B.Senthil** under my guidance during his postgraduate study period **2009-2012.**

This dissertation is submitted to **THE TAMILNADU Dr. M.G.R. MEDICAL UNIVERSITY,** in partial fulfillment for the degree of **MASTER OF DENTAL SURGERY, BRANCH IX – Oral Medicine and Radiology.**

It has not been submitted (partial or full) for the award of any other degree or diploma.

Guided By:

RAGAS DENTAL COLLEGE & HOSPITAL CHENNAI

Dr. Capt. S. Elangovan, M.D.S,
Professor
Department of Oral Medicine & Radiology
Ragas Dental College & Hospital
Chennai – 600 119.

Dr. (CAPT) S. ELANGOVAN, M.D.S.,
Prof. Oral Medicine & Radiology
RAGAS DENTAL COLLEGE & HOSPITAL,
2/102, East Coast Road Uthandi,
Chennai-600 119.

Dr. S. Shanmugam, M.D.S,
Professor and Head
Department of Oral Medicine & Radiology
Ragas Dental College & Hospital
Chennai – 600 119.

Dr. S. SHANMUGAM, M.D.S.
Prof. & H.O.D. Oral Medicine & Radiology
RAGAS DENTAL COLLEGE & HOSPITAL
2/102, East Coast Road Uthandi
Chennai - 600 119.

Dr. S. Ramachandran, M.D.S,
Principal,
Ragas Dental College & Hospital
Chennai – 600 119.

PRINCIPAL
RAGAS DENTAL COLLEGE AND HOSPITAL
UTHANDI, CHENNAI 600 119

Date: 02.11.2011

Place: Chennai - 600 119

ACKNOWLEDGEMENTS

Guru- Brahma Guru- Vishnu

Guru –Devo Maheshwara

Guru- Sakshat Parbrahama

Tasmai Sri Guruve Namah

"It is rightly said that Guru (preceptor) is greater than God; devotion to Preceptor is more meritorious than that to God. "

Words seem less to express my deep sense of gratitude to my postgraduate teacher, mentor, ***Dr.*** **S.** ***Shanmugam, M.D.S****., Professor and Head of the Department, Department of Oral Medicine and Radiology, Ragas Dental College and Hospital, for his valuable guidance, tireless pursuit for perfection, immense and constant support, encouragement throughout my post graduate curriculum. With all due respect I heart fully thank him for his perseverance in innovating me throughout my study period. Moreover, I thank him for all his kind help that have been conferred upon me without which this dissertation would not have come true.*

I take this opportunity to thank ***Dr. S. Ramachandran, M.D.S****., Principal, Ragas Dental College and Hospital, Chennai for the generous support rendered throughout my course.*

I express my deepest gratitude and profound indebtedness to ***Dr. Capt. S. Elangovan, M.D.S.,*** *Professor, Department of Oral Medicine*

and Radiology, Ragas Dental College and Hospital, Chennai for his valuable guidance.

I am extremely indebted to ***Dr.S.Kailasam, M.D.S.,*** *Professor, Department of Oral medicine and Radiology, Ragas Dental College and Hospital, Chennai and* ***Dr. Capt. S. Manoj Kumar, M.D.S.,*** *Professor, Department of Oral Medicine and Radiology, Ragas Dental College and Hospital, Chennai for their encouragement and support rendered throughout my course.*

My sincere thanks to ***Dr. P.E. Chandra Mouli, Dr. B. Anand, Dr.L.Aravind, Dr. P. Jagatheesh, Dr. Ramalakshmi*** *Senior Lecturers, for their encouragement and support rendered throughout my course.*

I also thank ***Mr.Ravanan,****M.Sc, M.Phil, PGDCA PhD, Clinical Epidemiologist, for his help and guidance in doing statistical analysis during my study.*

My sincere thanks to ***Dr. Janaki*** *MBBS, M.D.Medical Superintendent Voluntary Health Services, Adyar, Chennai for the generous support rendered throughout my study.*

My sincere thanks to ***Dr.C.V.Krishnaswami****,F.R.C.P.(E).D.T.M..&H (Edin) FAMS, Head of the Department,* ***Dr. Ramesh Chandrasekaran****, M.D.M.R.C.P.(U.K),D.T.M.&H.(London),* ***Dr. A.Srivatsa****, M.B.B.S,CCDE,*

***Dr.Saroja Srinivasan**,M.B.B.S, **Dr. ShobanaAnojan**, M.B.B.S **Mrs.S.lalitha**, Medical Secretary **Mrs.Thangamani**, **Mrs.Radha, Ms.R.Mary**, Staff Nurse, Department of Diabetology, Voluntary Health Service, Adyar for their encouragement and support rendered throughout my study.*

*My sincere thanks to **Dr. S.Bhaskaran,** M.D.**,** DNB (Neph), Mr**.S.Gandhi Kumar, Ms.Thangam, Ms. Manju**, Staff Nurse, Department Of Hemodialysis, Voluntary Health Service, Adyar for their encouragement and support rendered throughout my study.*

*My sincere thanks to **Ms. Fathima**, Dietician, Department Of Diabetology, Voluntary Health Service, Adyar for her encouragement and support rendered throughout my study.*

*I earnestly thank **Mrs. Kalyani** and **Mr. Balaji** of Department of Biochemistry, Voluntary Health Service,Adyar hospital, for performing necessary laboratory analysis during my study.*

*My sincere thanks to **Mr.A.Vinothkumar**, **Mr.N.Srinivasan**, Lab Technician, for performing necessary laboratory analysis during my study.*

I express my profound sense of gratitude to all the patients who participated in the study, and made this dissertation possible.

*I thank **Dr.P.MaheshKumar, Dr.M.Subha, Dr.R.Karthik** for their constant encouragement and support throughout my post graduate course.*

I also thank my batch mates ***Dr.R.Malavika, Dr.Ruchi Gera, Dr.S.Ramasubramanian****,* ***and Dr.S.Srividhya*** *and all my juniors for their unflinching help and support.*

I owe to my parents ***Thiru. R.Balasubramaniam*** *and* ***Tmt.B.Jothi*** *for what I am today .I also thank my brother* ***Dr. Kishore.B.Angrishi, Phd*** *(Germany) and sister-in-law* ***Tmt. Sujaritha.K.Angrishi,M.S*** *(Germany) for their innumerable sacrifice, love, understanding and support towards me.*

Above all I thank ***The Lord Almighty****, for without his grace nothing would have been possible.*

CONTENTS

LIST OF ABBREVIATIONS

S.NO	ABBREVIATION	EXPANSION
1.	DM	Diabetes Mellitus
2.	ESRD	End Stage Renal Disease
3.	CKD	Chronic Kidney Disease
4.	GFR	Glomerular Filtration Rate
5.	NESRD	Non End Stage Renal Disease
6.	NDD-CKD	Non-Dialysis Dependent Chronic Kidney Disease
7.	AGE	Advanced Glycation End-Products
8.	HD	Haemodialysis
9.	PD	Peritoneal Dialysis
10.	NTx	Renal transplantation
11.	mL	Milliliter
12.	mmol/L	Millimole per liter
13.	IDDM	Insulin Dependant Diabetes Mellitus
14.	NIDDM	Non Insulin Dependant Diabetes Mellitus
15.	WHO	World Health Organisation
16.	pH	Hydrogen ion concentration
17.	DKA	Diabetic Ketoacidosis
18.	DN	Diabetic Nephropathy
19.	US	United States
20.	BUN	Blood Urea Nitrogen
21.	KDOQI	Kidney Disease Outcomes Quality Initiative
22.	Ml/min	Milliter per minute
23.	m^2	Meter square
24.	Mg/dL	Milligram per decilitres
25.	MDRD	Modified Diet in Renal Disease Formula

26.	Cc	Creatinine clearance
27.	Serum Cr	Serum Creatinine
28.	Pcr	Plasma Creatinine
29.	SUN	Serum Urea Nitrogen
30.	Alb	Albumin
31.	µmol/L	Micro mole per liter
32.	mg%	Milligram percentage
33.	HBV	Hepatitis B Virus
34.	HCV	Hepatitis C Virus
35.	HIV	Human Immunodeficiency Virus
36.	DDAVP	1-Deamino-8-D-Arginine Vasopressin
37.	USRDS	United States Renal Data System
38.	IMSS	Instituto Mexicano Del Seguro Social
39.	REIN	Renal Epidemiology and Information Network
40.	g / kg / day	Gram per kilogram per day
41.	OFI	Oral Fungal Infection
42.	CRF	Chronic Renal Failure
43.	RRT	Renal Replacement Therapy
44.	HbA1C	Glycosylated Hemoglobin
45.	OC	Oral Candidiasis
46.	GH	Gingival Hyperplasia
47.	CHL	Compatible with Hairy Leukoplakia
48.	ST	Saburral Tongue
49.	SU	Sulfonylurea

LIST OF TABLES

LIST OF GRAPHS

LIST OF FIGURES

End Stage Renal Disease (ESRD) is defined as the near failure or failure of the kidneys to perform their normal functions like, excretion; maintenance of acid-base, fluid and electrolyte balance and the synthesis of hormones such as erythropoietin and renin[1].

End Stage Renal Disease (ESRD) is the final stage of chronic kidney disease (CKD) and ESRD usually results from a progressive and irreversible loss of renal function and is defined by a glomerular filtration rate (GFR) of less than 15 ml/min. In Chronic Kidney Disease (CKD), the pre-End stage renal disease is termed as Non End Stage Renal Disease (NESRD) or Non-dialysis dependent CKD (NDD-CKD)[1]

The most common causes of ESRD are chronic hypertension, glomerulonephritis, polycystic kidney disease, renovascular disease and diabetes mellitus.[2]

The term "Diabetes Mellitus" is used to identify a group of disorders characterized by elevated levels of glucose in the blood. This elevation is the result of a deficiency in insulin secretion or an increased cellular resistance to the actions of insulin, leading to a variety of metabolic abnormalities involving carbohydrates, fats and proteins[3].

A number of pathological mechanisms related to elevated levels of glucose in the blood have been defined, including the activation of the sorbitol pathway, the formation of advanced glycation end-products (AGEs), the damaging effect of oxidative stress and altered lipid metabolism. These mechanisms have been associated with classical clinical

complications of Diabetes Mellitus such as retinopathy, nephropathy, neuropathy, macrovascular disease and poor wound healing[4,5].

Diabetes mellitus is a major health problem in today's society as the number of patients with Diabetes is growing continuously. Globally, by the year 2010 more than half of the world's diabetics will be Asians[6,7].

Patients with ESRD can rely on kidney replacement therapeutic modalities such as haemodialysis (HD), peritoneal dialysis (PD) or renal transplantation (NTx). Although kidney replacement therapies have proven to be successful in prolonging the life expectancy of ESRD patients, several limitations and long-term complications exist[8,9] .Since the majority of haemodialysis patients has no residual urine output, they have to maintain a fluid restricted diet to prevent fluid overload and are thus allowed to consume only approximately 500 mL per day[10] .If patients do not adhere to the restriction in fluid intake, (chronic) fluid overload may occur, which can result in hypertension, acute pulmonary oedema, congestive heart failure and consequently death[11,12] .

As Diabetic Nephropathy progresses to End-Stage Renal Disease (ESRD) and impose enormous medical, economic and social costs on both the patient and the health care system, it deserves greater social concern. In fact, Diabetic Nephropathy has become the major cause of ESRD[13]

The population with End-Stage Renal Disease (ESRD) in the United States is composed of more than 200,000 patients who undergo dialysis and 70,000 patients with functioning kidney transplants. With the prevalence of

ESRD growing at a rate between 7 to 9 percent per year, it is projected that there will be more than 350,000 such patients by the year 2010.[14]

Hence it is felt the need to identify the disease at an early stage based on the oral manifestation in NESRD- Diabetes Mellitus patients to avoid the complication and for suitable treatment, so a study has been undertaken to detect the oral manifestation in already diagnosed ESRD-Diabetes Mellitus and NESRD-Diabetes Mellitus patient and to compare them in both the conditions.

AIM OF THE STUDY:

To assess oral signs, symptoms and oral lesions type and prevalence, in diabetic patients with End Stage Renal Disease (ESRD-DM) and compare them with analogous findings in Non-End Stage Renal Disease (NESRD-DM) group.

OBJECTIVE OF THE STUDY:

1. To compare the oral signs, symptoms and oral lesions type and prevalence in diabetic patients with End Stage Renal Disease (ESRD-DM) and with Non-End Stage Renal Disease (NESRD-DM) group
2. To analyze the possible association between oral manifestation as well as with relevant laboratory findings

DIABETES MELLITUS

DEFINITION

Diabetes Mellitus is a clinical syndrome comprising a heterogeneous group of metabolic diseases that are characterized by chronic hyperglycemia and disturbances in carbohydrate, fat and protein metabolism secondary to defects in insulin secretion, insulin action or both.[15]

The World Health Organization (WHO) has considered Diabetes Mellitus a public health problem since 1975. Therefore, it is necessary that health care professionals become interested on the disease in order to provide an appropriate treatment to these patients in the different fields of knowledge[15].

Diabetes is a dangerous disease since the patient's and healthcare promoter's negligence may impair the patient's quality of life and even lead the patient to death. Diabetes is a disease in which the insulin's regulatory activity is defective. This can be a result of decreased amount of insulin that should be secreted, total absence of insulin secretion or the production of antibodies against insulin causing its destruction before it can act in the different areas of the body. In the first two cases there is degeneration or inactivation of beta cells of the Langerhans islets which produce insulin. In the last case, the amount of insulin secreted may be normal but it does not reach its destination[15].

WHO-Criteria for the Diagnosis of Diabetes:

1. Symptoms of Diabetes plus casual venous plasma glucose more than or equal to11·1 mmol/l. Casual is defined as any time of day without regard to time since last meal. The classic symptoms of diabetes include polyuria, polydipsia, polyphagia and unexplained weight loss
2. Fasting plasma glucose more than or equal to 7·0 mmol/l or whole blood than or equal to 6·1 mmol/l. Fasting is defined as no calorie intake for at least 8 hours
3. Two hour plasma glucose more than or equal to 11·1 mmol/l during oral glucose tolerance test using 75 g glucose load

In the absence of symptoms, these criteria should be confirmed by repeat testing on a different day. If the fasting or random values are not diagnostic, the 2 hour value post-glucose load should be used

Note:

Fasting plasma glucose less than 6·1 mmol/l—normal

Fasting plasma glucose more than or equal to 6·1 and less than 7·0 mmol/l —impaired fasting blood glucose

Fasting plasma glucose more than or equal to 7·0 mmol/l—provisional diagnosis of Diabetes; the diagnosis must be confirmed[5]

CLASSIFICATION OF DIABETES MELLITUS

In 1997, the American Association of Diabetes proposed a classification system for diabetes based on its etiology. Therefore, diabetes is currently classified as:

- Type 1 or Juvenile Diabetes or IDDM –Insulin Dependant Diabetes Mellitus and
- Type 2 or Acquired Diabetes or NIDDM - Non Insulin Dependant Diabetes Mellitus

Type 1 Diabetes was defined as onset of Diabetes before 35 years of age and insulin treatment initiated within one year of diagnosis of Diabetes.

Type 1 Diabetes it appears in the first or second decade of life; it is caused by the destruction of pancreatic beta cells, which can be caused by a viral or an autoimmune process leading to a blockade in the production of insulin[16].

On the other hand, Type 2 Diabetes is the result of an abnormality that can occur both at the molecular level of insulin and at the cellular level of insulin receptors[15].

Rivera[17] suggested that the appearance of Alzheimer disease may be associated with a new type of Diabetes named Type 3 Diabetes. Although the pancreas is the main organ responsible for insulin secretion, the fall of insulin levels in the brain causes the so-called Type 3 Diabetes. In this study it was found that the brain produces a small amount of insulin and proteins.

The fact that there is an appropriate level of insulin as well as the correct activity of the receptors is described as vital for the cell survival in the brain.

It is estimated that there are about 170 million people with Diabetes Mellitus in the world and approximately 10 million in Brazil. Of those, approximately 50% do not know they have the disease. According to the WHO, about 7% of the world adult population has Diabetes. In the state of São Paulo this rate reaches 9.6%[15].

Paradella et al[18] stated that the main symptoms of the patient with Diabetes Mellitus are polydipsia, polyuria-nocturia, polydipsia associated with xerostomia, polyphagia, vulvar pruritus, rapid weight loss, even with a balanced diet. Visual changes (such as blurred vision), somnolence, pain, cramps, fatigue, tingling and numbness of lower limbs, asthenia, organ deficiency, indisposition to work, discouragement, generalized physical and mental tiredness, ketoacidosis and fruit breath are also observed.

To meet cellular energy needs, fat is broken down through lipolysis, releasing glycerol and free fatty acids. Glycerol is converted to glucose for cellular use. Fatty acids are converted to ketones, resulting in increased ketone levels in body fluids and decreased hydrogen ion concentration (pH). Ketones are excreted in the urine, accompanied by large amounts of water. The accumulation of ketones in body fluids, decreased pH, electrolyte loss and dehydration from excessive urination, and alterations in the bicarbonate buffer system result in Diabetic Ketoacidosis (DKA).

Diabetic ketoacidosis (DKA) is a potentially life-threatening complication in patients with Diabetes Mellitus. It happens predominantly in those with Type 1 Diabetes, but it can occur in those with Type 2 Diabetes under certain circumstances. DKA results from a shortage of insulin; in response the body switches to burning fatty acids and producing acidic ketone bodies that cause most of the symptoms and complications[19].

Untreated DKA can result in coma or death. Many patients with Type 1 Diabetes are initially diagnosed with the disease following a hospital admission for DKA. In a known Diabetic patient, periods of stress or infection may precipitate DKA. More often, however, DKA results from poor daily glycemic control. Patients who remain severely hyperglycemic for several days or longer due to inadequate insulin administration or excessive glucose intake are prone to develop DKA.[20]

Blood samples are usually taken to measure urea and creatinine (measures of kidney function, which may be impaired in DKA as a result of dehydration) and electrolytes[19].

A 2006 American Diabetes Association statement (for adults) categorizes DKA into one of three stages of severity[19]:

- Mild: blood pH mildly decreased to between 7.25 and 7.30 (normal 7.35–7.45), serum bicarbonate decreased to 15–18 mmol/l (normal above 20), the patient is alert

- Moderate: pH 7.00–7.25, bicarbonate 10–15, mild drowsiness may be present
- Severe: pH below 7.00, bicarbonate below 10, stupor or coma may occur

Type 2 Diabetes Mellitus patient taking metformin may develop lactic acidosis as a side-effect of their medication[19]

In regards to the specific role of Otorhinolaryngology, **Scherer and Lobo**[21] noticed irritative vestibular disorder in patients with Type I Diabetes. **Maia and Campos**[22] state that there is evidence that Diabetes Mellitus may cause hearing loss.

REVIEW OF LITERATURE FOR DIABETES MELLITUS

According to **Tommasi**[23], the most common oral manifestations in Diabetic patients include xerostomia, burning and eventual erythema, ulcerations, pharyngeal infections caused by Candida albicans, cheilitis, lichen planus, tumefaction of salivary glands, gingival problems, periodontal problems, abscesses and marked loss of alveolar bone, although none of them is a pathognomonic lesion. In the patient with uncontrolled Diabetes, a decreased response to infection (bacterial, fungal and viral) is observed, due to the hyperglycemia and ketoacidosis that changes the phagocytosis of macrophages and the chemotaxis of polymorphonuclear neutrophils. The patient with controlled Diabetes without vascular disease does not present increased rates of infection since a good control of the

disease reduces the likelihood of infection to a minimum, and repair does not seem to be very different from that seen in the Non-Diabetic patient. In 1993, the WHO included the periodontal disease as a classic complication of Diabetes.

Löe[3] proposed that periodontal disease was the sixth complication of Diabetes Mellitus.

Ship[5] listed dental caries, salivary dysfunction, oral mucosal diseases, oral infections such as candidiasis, taste and other neurosensory disorders. Furthermore, a reduction in salivary flow has been reported in people with Diabetes who have neuropathy and diminished salivary flow is a risk factor for dental caries.

Moore PA et al[24] found that Dry mouth, or xerostomia, has been reported in people with Diabetes Mellitus. Salivary flow may be affected by a variety of conditions, including the use of prescription medications and increasing age, and it appears to be affected by the degree of neuropathy and subjective feelings of mouth dryness that may accompany thirst. These variables are relevant for adults with Diabetes Mellitus. Therefore, although no definitive association of Diabetes and reduced salivary flow has been identified, this complication has been reported in people with Diabetes. A number of types of oral mucosal lesions, including lichen planus and recurrent aphthous stomatitis have been reported in people with Diabetes Mellitus. Not all study results have showed this association, and these are

relatively common disorders that often are observed in patients who do not have Diabetes.

Guggenheimer J et al[25], conducted a cross-sectional study, compared the prevalence of candidiasis in 405 subjects with IDDM and 268 nondiabetic control subjects and found that oral candidiasis has been a more consistent finding in patients with Diabetes (15.1%). Candidiasis is a manifestation of an immunocompromised state, and a reduction in salivary flow is another risk factor for oral candidiasis (P <0.0001).

Taste disturbances have been reported in patients with Diabetes Mellitus but all investigators have not observed this finding. Although patients with Diabetes who receive hemodialysis have been reported to have altered taste it is a complex symptom, and it may be related to salivary flow and changes in food intake associated with disease management. Other neurosensory disorders of the oral and perioral tissues, including burning mouth syndrome and dysphagia, have been reported in patients with Diabetes

Tommasi[23]**, Sousa et al**[26]**, Yuli Muller and Yuraima**[27], observed a high frequency of Candida infections in patients with Diabetes Mellitus. The clinical manifestations and the oral symptoms of Diabetic patients may vary from a minimum to a more aggressive stage and depend on the type of existing hyperglycemic abnormality, of treatment control and the time elapsed since the diagnosis of the disease.

Belmiro Cavalcanti do Egito Vasconcelos et al[28] conducted a study on 30 patients diagnosed as diabetes, 9 (30%) were males and 21 (70%)

females. Of the studied patients, 40% were below 60 years of age, and 60% were older than 60 years. Thirteen different types of mucosal alterations were diagnosed. Tongue varicose veins (36.6%) and candidiasis (27.02%) were the most prevalent. Such alterations can be associated with the fact that these conditions are commonly found in senile patients and are also associated with prolonged wear of dentures. Xerostomia was diagnosed in only 1 (3.33%) patient,

DIABETIC NEPHROPATHY

DEFINITION

Diabetic Nephropathy is a clinical syndrome characterized by persistent albuminuria, elevated arterial blood pressure, a ceaseless decline in glomerular filtration rate (GFR) and a high risk of cardiovascular morbidity and mortality[29,30]. It is progressive and usually irreversible.

The natural history of Diabetic Nephropathy is complex and most changes in the kidneys are currently undetectable in clinical practices[31]. Renal involvement in Diabetes is presumed to be resulted from the interplay between several metabolic and homodynamic processes [32,33].

In brief, Diabetic Nephropathy is morphologically featured by either diffuse or selective expansion of the mesangial matrix, which leads to obliteration of capillary lumen and loss of filtration surface area. Clinically, microalbuminuria is the hallmark of Diabetic Nephropathy and accounts for subsequent progressive renal dysfunction among Diabetes people[34]. With no therapeutic intervention, serum creatinine levels continue to climb and

patients go on to develop ESRD eventually[35]. Patients with Diabetes with the highest GFR early in the course of their disease are found very likely to develop Diabetic Nephropathy[36,37] . It is mainly seen in Type 1 Diabetic patients, but also probably noted in those with Type 2.

Diabetic Nephropathy is the most common cause of End-Stage Renal Disease requiring dialysis in the US [38]. The incidence of Diabetic Nephropathy in this country has increased substantially over the past few years. Advanced Diabetic Nephropathy is also the leading cause of glomerulosclerosis and End-Stage Renal Disease worldwide [39]. Between 20% and 40% of patients with Diabetes ultimately develop nephropathy, although the reason why not all patients with Diabetes develop this complication is unknown.[40]

The natural history of Diabetic Nephropathy differs according to the type of Diabetes and whether microalbuminuria (defined as more than 30 mg but less than 300 mg albumin in the urine per day) is present. If untreated, 80% of people who have Type 1 Diabetes and microalbuminuria will progress to overt nephropathy (i.e. proteinuria characterized by more than 300 mg albumin excreted daily), whereas only 20-40% of those with Type 2 Diabetes over a period of 15 years will progress[40].

Nielsen[41] demonstrated more than a decade ago, a clear, early predictor of disease progression is increasing systolic blood pressure, even within the prehypertensive range. Among patients who have Type 1

Diabetes with nephropathy and hypertension, 50% will go on to develop End-Stage Renal Disease within a decade.

Mortality among dialysis patients with Diabetes is 22% higher in the first year following the initiation of dialysis and 15% higher at 5 years than that among dialysis patients without Diabetes [42].

Diabetic Nephropathy has several distinct phases of development. Functional changes occur in the nephron at the level of the glomerulus, including glomerular hyper filtration and hyper perfusion, before the onset of any measurable clinical changes. Subsequently, thickening of the glomerular basement membrane, glomerular hypertrophy, and mesangial expansion take place[43].

Steinke[44] demonstrated that individuals with Type 1 Diabetes and microalbuminuria in whom these histological alterations were detected were destined to progress to overt nephropathy.

Microalbuminuria, however, has a variable course; its progression to macroalbuminuria (> 300 mg per day) is unpredictable and does not always lead to development of nephropathy[35]. Moreover, the rate of kidney function decline after the development of nephropathy is highly variable between patients and is influenced by additional factors, including blood pressure and glycemic control.

The Progression of Diabetic Nephropathy occurs in five stages[40]

The Stage 1 Diabetic Nephropathy (normal) is characterized by:

- Albuminuria is less than 20 mg in 24 hours
- Glomerular filteration rate is high or normal hyperfilteration
- Serum Creatinine level is 60-100 µmol/l
- The blood pressure is normal
- There is no clinical signs

The Stage 2 Diabetic Nephropathy is characterized by:

- Albuminuria is between 20- 300 mg in 24 hours
- Glomerular filteration rate is normal or high
- Serum Creatinine level is 60-120 µmol/l
- The blood pressure is slightly increased
- There is no clinical signs
- This stage is called as Incipient Diabetic Nephropathy

The stage 3 Diabetic Nephropathy is characterized by:

- Albuminuria is more than or equal to 300 mg in 24 hours
- Glomerular filteration rate is normal or decreased
- Serum creatinine level is 80-120 µmol/l
- The blood pressure is increased
- The clinical signs includes anaemia with or without oedema
- This stage is called as Persistent Diabetic Nephropathy

The stage 4 Diabetic Nephropathy is characterized by:

- Albuminuria is more than or equal to 300 mg in 24 hours
- Glomerular filteration rate is decreased
- Serum creatinine level is 120- 400 µmol/l
- The blood pressure is increased
- The clinical signs includes anaemia with or without oedema
- This stage is also called as Clinical Diabetic Nephropathy

The stage 5 Diabetic Nephropathy is characterized by:

- Albuminuria is more than or equal to 300 mg in 24 hours
- Glomerular filteration rate is greatly decreased
- Serum Creatinine level is very high ,more than 400 µmol/l
- The blood pressure is increased
- The clinical signs includes anaemia with or without oedema, and uremic symptoms can also be seen
- This stage is also called as End Stage Diabetic Nephropathy[40]

The accumulation of creatinine and blood urea nitrogen (BUN), waste products in the blood is called Azotemia. Early detection of Azotemia is vital to preserve kidney function and to delay or prevent ESRD. According to the National Institute of Diabetes & Digestive & Kidney Diseases, individuals with Type 2 Diabetes may remain in this stage for several years[38].

Stage V, or ESRD, is when the kidneys fail to function, the GFR severely decreases, and hypertension continues to worsen. During this final stage, the kidneys cannot excrete toxins; maintain fluid, pH, and electrolyte balances; or secrete important hormones (renin, vitamin D, and erythropoietin) [45].

DEFINITION AND CLASSIFICATION OF RENAL DISEASE

In 2002, the National Kidney Foundation's Kidney Disease Outcomes Quality Initiative (KDOQI) introduced a conceptual model for the definition and classification of chronic kidney disease.[1] The model included antecedents associated with increased risk for development of chronic kidney disease, earlier stages of disease that could progress to later stages or lead to complications, and kidney failure as the end stage . Chronic kidney disease was defined based on the presence of kidney damage or glomerular filtration rate (GFR <60 ml/min per 1.73m^2) for less than or equal to 3 months, irrespective of cause, and was classified into five stages based on the level of GFR.[1]

1. Stage 1 condition characterized by kidney damage with normal or increased GFR i.e., GFR $\geq$ 90 mL/min/1.73m^2
2. Stage 2 condition characterized by kidney damage with mild decrease in GFR i.e., GFR is between 60- 89 mL/min/1.73m^2
3. Stage 3 condition characterized by kidney damage with moderate decrease in GFR i.e., GFR is between 30-55 mL/min/1.73m^2

4. Stage 4 condition characterized by kidney damage with severe decreased in GFR i.e., GFR is between 15-25 mL/min/1.73m^2
5. Stage 5 condition is characterized by kidney failure i.e., GFR is < 15 mL/min/1.73m^2

End-Stage Renal Disease is defined as stage 5 of chronic kidney disease i.e., it appears when GFR decreases to <5-10 mL/min/1.73 m^2. At this point the patient is seriously ill, with pronounced uremic symptoms. High blood pressure, weight loss, anemia, neuropathy and osteodystrophy indicate that the patient needs to start dialysis treatment. Normal change in GFR values is usually measured by creatinine clearance (CC), which gives an acceptable approximation of the value of GFR[46].

The glomerular filtration rate (GFR) is traditionally considered the best overall index of renal function in health and disease. Because GFR is difficult to measure in clinical practice, most clinicians estimate the GFR from the serum creatinine concentration[47].

Serum and urine creatinine were measured by using a kinetic alkaline picrate assay or Serum creatinine was determined at a central laboratory by a kinetic Jaffe reaction using a Hitachi 911 E analyzer (Boehringer Mannheim, Mannheim, Germany),with a normal range in serum of 62 to 124 mmol/L (0.7 to 1.4 mg/dL)[47] .

At any given GFR, the serum creatinine concentration is significantly higher in men than in women and in black persons than in white persons

Patients with a serum creatinine concentration of 124 mmol/L (1.4 mg/dL), a value within the normal range in many clinical laboratories, would have renal insufficiency, as defined by serum creatinine concentration, creatinine clearance, or GFR less than two standard deviations below the normal range[47]. Patients with a serum creatinine concentration of 354 mmol/L (4.0 mg/dL) would be approaching end-stage renal disease, as defined by a GFR less than 10 mL/min per 1.73 m^2.

Normal values of serum Cr are 0.5-1.4 mg/dl; in patients with renal insufficiency, Cr will be of 1.5 mg/dl or more. Plasmatic creatinine can be related to creatinine clearance using several formulas, such as Cockcroft-Gault or MDRD (modified diet in renal disease formula)[1].

Cockcroft-Gault equation estimates creatinine clearance (Cc),

Where Pcr is plasma creatinine:

Creatinine clearance (Cc) =

$$\left[\frac{(140 - \text{age}) \times \text{weight (kgs)}}{72 \text{ x Pcr}}\right]$$

(multiply by 0.85 for women)

MDRD (modified diet in renal disease formula) estimates GFR

(Where Pcr is plasma creatinine, SUN is serum urea nitrogen, and Alb is albumin):

$GFR = 170 \text{ X } (Pcr)^{-0.999} \text{ X } (Age)^{-0.176} \text{ X } (SUN)^{-0.017} (Alb)^{+0.318} \text{ X } (0.762 \text{ if patient is female}) \text{ X } (1.180 \text{ if patient is black})$

Presently, the classification of chronic renal disease is guided by the National Kidney Foundation's K-DOQI guides (The National Kidney Foundation Kidney Disease Outcomes Quality Initiative) of 2002, which include the following situations:

- Kidney damage during at least 3 months with or without a decrease in glomerular filtration rate
- Filtration rate < 60 ml/min/1.73 m^2 during more than 2 months with or without kidney damage [1].

End-Stage Renal Disease (ESRD) is that stage of kidney impairment which is irreversible, cannot be controlled by conservative management alone, and requires dialysis or kidney transplantation to maintain life.

There are two types of kidney failure[1]: acute and chronic.

1. Acute kidney failure is a temporary decline in kidney function that can most often be corrected.
2. Chronic kidney failure, on the other hand, is a permanent condition, meaning that once it occurs, the kidneys cannot be made to function again.

Chronic kidney failure may be the result of heredity, as with polycystic kidney disease, or may be caused by prolonged medical conditions, such as high blood pressure or diabetes. Persons with chronic renal failure are referred to as having end-stage renal disease[1].

THE DEVELOPMENT OF KIDNEY DISEASE

For many years glomerulonephritis has been the most common disease for patients on dialysis. The incidence of Diabetes Mellitus (DM) is now increasing fast and will soon result in the most common diagnosis in dialysis treatment. When a systemic disease affects the kidneys it is referred to as secondary.

Patients with diabetes are by far the largest group; 25-45% develops a diabetic nephropathy. Irrespective of whether the diabetes is type 1 or 2, a majority of the individuals can be detected early because of microalbuminuria, which is a sign of poor prognosis. Consequently glomerulosclerosis and atherosclerosis appear and GFR gradually declines, leading to end-stage renal disease.[46]

About one-fifth of diabetic patients develop End-Stage Renal Disease (ESRD) during their lifetime. Diabetes is currently the major cause of renal failure requiring dialysis treatment or renal transplantation in the US, Europe, and Japan, and its incidence is rapidly increasing in all countries, leading to the definition of Diabetic Nephropathy as 'a medical catastrophe of worldwide dimensions' [48]

Although rates of dialysis initiation and of renal transplantation are readily available in Europe and the US, this information only approximates the true incidence and prevalence of ESRD, because the definition of ESRD and the criteria for dialysis initiation might differ across countries.[48]

RISK FACTORS FOR DEVELOPMENT OF CKD[49]

- Underlying disease:
  - Hypertension
  - Diabetes
  - Dyslipidemia
- Lifestyle factors:
  - Tobacco
  - Inactivity
- Family history
- Aging
- Prenatal factors:
  - Maternal Diabetes Mellitus
  - Low birth weight
  - Small-for-gestational-age status

Chih-Cheng Hsu et al[50] performed a study that included 6,001 subjects, and found that the prevalence of CKD stages 3 to 5 in Taiwan is 6.9% (95% confidence interval, 4.4 to 9.4). Awareness rates for CKD in Taiwan are low: 8.0% for individuals with stage 3, 25.0% for those with stage 4, and 71.4% for those with stage 5. Awareness rate is related closely to serum creatinine level: those with creatinine levels greater than 1.6 mg/dL (>141 μmol/L) are more likely to be informed of having a kidney disease

Sanjay Kumar Agarwal et al[51] stated that a Serum Creatinine >1.8mg% as renal failure. They suggested a repeat test for Serum Creatinine which was done after 8–12 weeks to confirm chronicity of renal failure. If it was >1.8mg% after 3 months in the absence of reversible factors, CRF was diagnosed.

MEDICAL MANAGEMENT:

Use of Antihyperglycemic Agents in Patients with Renal Failure[52]

Metformin is contraindicated in renal failure because of the associated risk for lactic acidosis. It can be used at low dosages up to a creatinine clearance of 30 to 60 ml/min and should be avoided with clearances less than 30. Although the metabolism of thiazolidinediones is unaffected by renal failure, they must be used with caution in this context because of their volume retaining effect with a risk for heart failure.

The sulfonylureas (glyburide, gliclazide, glipizide, glibenclamide, tolbutamide, and chlorpropamide) have increased potency as the renal function decreases and are contraindicated in severe renal failure. The nonsulfonylurea insulin secretagogues repaglinide and nateglinide can be used in renal failure without dose adjustments. α-Glucosidase inhibitors (acarbose and miglitol) are contraindicated in renal failure[52].

a) Patient with renal disease in conservative medical treatment[52]:

For the dental treatment of these patients, good communication with their nephrologist is highly recommended, in order to be aware of the stage of the pathology suffered and the treatment prescribed. Before any invasive dental

procedure, possible hematologic problem in the patient should be studied. It is essential to remove any infective foci as soon as possible. Due to the frequent hypertension, blood pressure should be monitored during the procedures. When prescribing drugs, those that are nephrotoxic must be avoided (tetracyclines, aminoglycosides), some of them need a dose adjustment (as previously detailed). Apart from these considerations, no more exceptional measures must be performed[52].

b) Patient with renal disease in peritoneal dialysis:

In cases of peritoneal dialysis, a catheter placed in the abdominal wall and inserted in the peritoneum achieves access to the body, in order to remove nitrogen and other metabolic toxic products; through this, a hypertonic glucosated solution is introduced. Peritoneal membrane of the patient filters blood waste products by means of an osmotic mechanism, so they would be transferred to the electrolytic solution and then evacuated to the exterior [53]. This form of dialysis can be performed at home, but must be done every day. These patients do not require special measures with regard to dental treatment, apart from the considerations already mentioned [54].

c) Patient with renal disease in hemodialysis:

In hemodialysis, the filtering process is carried out by a machine (dialyzer), outside the patient's body. Most of these patients receive this treatment 3 times a week. In order to take the blood out of the body and to return it, it is necessary to have a vascular access. Permanent access is obtained by surgically connecting an artery to a vein, using a blood vessel (arteriovenous

fistula) or a synthetic bridge (arteriovenous graft).[55] A special solution (dyalisate) corrects the chemical disturbances and impurities of the blood, which is then introduced into the body . During the process of hemodialysis, the patient's blood is anticoagulated with heparin to facilitate blood transit. For this reason, dental treatments with a risk of bleeding must not be performed the day of hemodialysis[56]. If an emergency dental treatment must be performed, protamine sulphate (heparin antagonist) can be administered to block the anticoagulant effect. However, bleeding tendency would be still present due to platelet dysfunction and anemia, so usual hemostatic measures must be carried out[57]. There is a risk of infection because of the vascular access, and of transmission of HBV, HCV and HIV, so this must be confirmed in patients receiving hemodialysis[58].

Capillary blood glucose and the insulin flow rate were recorded every 30 min. Five milliliters of venous blood were collected for the measurement of blood urea nitrogen and serum creatinine immediately before and after the session. Blood urea nitrogen and serum creatinine were determined on venous blood samples collected immediately before and at the end of the hemodialysis session using colorimetric methods. The quality of dialysis was calculated as the logarithm of the ratio of serum urea concentrations at the beginning and at the end of hemodialysis[59].

Creatinine failure was defined as a serum creatinine value greater than 2.0 mg/dl[60]

Dental Management of the patient receiving hemodialysis patients depends on the stage of dental procedure [61]

1. Before treatment
    - Determine dialysis schedule and treat on the day after dialysis.
    - Consult with patient's nephrologist for recent laboratory tests and discussion of antibiotic prophylaxis.
    - Identify arm with vascular access and type; notate in chart and avoid taking blood pressure measurement/injection of medication on this arm.
    - Evaluate patient for hypertension/hypotension.
    - Institute preoperative hemostatic aids (DDAVP (1-deamino-8-D-arginine vasopressin, conjugated estrogen)) when appropriate.
    - Determine underlying cause of renal failure (underlying disease may affect provision of care).
    - Obtain routine annual dental radiographs to establish presence and follow manifestations of renal osteodystrophy.
    - Consider routine serology for HBV, HCV, and HIV antibody.
    - Consider antibiotic prophylaxis when appropriate.
    - Consider sedative premedication for patients with hypertension[61].
2. During treatment
    - Perform a thorough history and physical examination for presence of oral manifestations.

- Aggressively eliminate potential sources of infection/bacteremia.
- Use adjunctive hemostatic aids during oral/periodontal surgical procedures.
- Maintain patient in a comfortable uncramped position in the dental chair.
- Allow patient to walk or stand intermittently during long procedures.[61]

3. After treatment
   - Use postsurgical hemostatic agents.
   - Encourage meticulous home care.
   - Institute therapy for xerostomia when appropriate.
   - Consider use of postoperative antibiotics for traumatic procedures.
   - Avoid use of respiratory-depressant drugs in presence of severe anaemia.
   - Adjust dosages of postoperative medications according to extent of renal failure.
   - Ensure routine recall maintenance.[61]

a) Renal transplant patient

These patients are immunosuppressed by medication. Maintenance of a proper oral health is especially important as oral infections in transplant patients can contribute to its morbidity or even rejection [55]. They are usually receiving a treatment of corticosteroids, calcineurin inhibitors (tacrolimus)

and inhibitors of lymphocyte proliferation (azathioprine, mycophenolate mophetil)[49]. Long- standing treatment with high doses of corticosteroids produces an adrenal function suppression, which predisposes the patient to suffer an acute complication, adrenal crisis, when exposed to stressful situations like disease, infection, surgery. Furthermore, this chronic excess of corticosteroids can cause Cushing's syndrome[62]. To minimize the risk of suffering an adrenal crisis in patients taking high doses of corticosteroids who are undergoing a surgical procedure, they should take a double dose of corticosteroids the day of the surgery. This supplement will not be necessary if the patient is being treated with low doses (less than 7.5 mg of prednisolone) or if the patient is undergoing a conservative treatment[52]. However, the risk of developing an adrenal crisis after oral surgery procedures under local anesthesia is very low and the majority of dental treatments can be carried out without prescribing a supplement of corticosteroids [62].

REVIEW OF LITERATURE FOR ESRD AND NESRD IN DIABETES MELLITUS

End Stage Renal Disease (ESRD) is the final syndrome for several primary renal diseases, and systemic diseases with renal involvement, causing kidney function loss. ESRD manifestations involve virtually every system, in a clinical condition known as uremic syndrome, characterized by a profound alteration of water, electrolyte, and acid-base homeostasis, as

well as retention of uremic toxins normally eliminated through urine, especially protein catabolism nitrogen waste products[63]. The condition is incompatible with life, unless the patient starts chronic dialysis treatment or kidney transplantation.

ESRD incidence and prevalence are increasing, as shown in consecutive United States Renal Data System (USRDS) annual data reports. All age groups are affected, but ESRD is predominantly an adult disease. ESRD cause was Diabetes in 44.8% of incident USA cases in 2003. In that same report, chronic dialysis patient's prevalence was 1,496 per million, and median age at dialysis start increased from 52.8 years in 1980, to 62.7 years in 2003, reflecting improved kidney disease medical care [64].

An ESRD prevalence study on Instituto Mexicano del Seguro Social (IMSS)-affiliated adults (>18 yr), estimated 1.142 persons with creatinine clearance levels <15 ml/min per million adult affiliates [65]. That level of renal function damage does already, or will soon, need dialysis treatment. Another study on IMSS affiliates found Diabetes Mellitus as the cause of ESRD in 41.1% of incident cases. ESRD mortality is increasing in Mexico; being now 9th cause for women and 10th for men .Diabetes Mellitus is an important risk factor for ESRD[66].

Chi-yuan Hsu et al[67] found The prevalence of chronic renal insufficiency among older adults was 10-fold that of younger individuals . However, younger individuals with chronic renal insufficiency were about 3-fold more likely to progress to ESRD, presumably because of a decreased

risk for competing mortality Diabetic patients with chronic renal insufficiency were also about 3-fold more likely to progress to ESRD than Non-Diabetic patients with chronic renal insufficiency. For each 1000 cases of chronic renal insufficiency in 1991, 38 cases of ESRD developed among Diabetic patients in 1996 compared with 11 cases among Non-Diabetic patients

In the **REIN registry**, among ESRD patients aged more than or equal to 45 years with associated diabetes, only 58.0% had Type 1 Diabetes in the year 2006 [68]

Zohreh Hajheydari and Atieh Makhlough[69] found that the patients with chronic renal insufficiency were 43 (42.6%) women and 58 (57.4%) men with a mean age of 50.0 ± 12.3 years. The duration of hemodialysis was 36.0 ± 11.0 months. Hair, mucous membrane, and nail problems were present in 37.6%, 23.8%, and 43.6% of the patients, respectively. There was a significant association of the number of cutaneous manifestations with the age of the patients (P = .001). Cutaneous and mucosal disorders are of the common problems in patients on long-term hemodialysis. The most common oral mucosal problem was furred tongue (7.9%). Of the nail disorders, nail bed paleness (16.8%) was the most common.

Francois Madore et Al[70] found variables of nutritional status (serum albumin and creatinine concentration), and the dose of dialysis (urea reduction ratio) were found to be significantly associated with hemoglobin

concentration (P < 0.001). Age, race, and gender were also found to be significantly associated with haemoglobin concentrations (P < 0.001).

Gall et al[71] conducted a prospective observational study involving 176 patients with Type-2 Diabetes, and found that males had a 2.6 times greater risk of developing incipient or overt nephropathy.

Choy BY et al[72] found that for ESRD patients, the male/female ratio was reported to be about 1:1 for Diabetes Patients

Olugbenga. E. Ayodele et al[73] found that Smoking causes a substantial increase in the risk of both micro- and macrovascular diseases in Diabetes. Smoking is an independent risk factor for the development of Diabetic Nephropathy and is associated with an accelerated loss of renal function, an increased risk for ESRD, and decreased survival on commencement of dialysis. Loss of renal function is slower in those who stopped smoking. Cessation of smoking alone may reduce the risk of progression by 30% in patients with Type-2 Diabetes.

Jorge l Gross et al[74] found in 108 patients, that dietary protein restriction slowed the progression of Diabetic Nephropathy in patients with Type 1 Diabetes. More recently, a 4-year randomized controlled trial in 82 patients with Type 1 Diabetes with progressive Diabetic Nephropathy showed that a moderately low–protein diet (0.9 g / kg / day) reduced the risk of end-stage renal disease or death by 76%.

Thorman R, Neovius M and Hylander B[75] conducted a study on 101 patients and found 43 (42.6%) were women and 58 (57.4%) were men

with a mean age of 50.0 ± 12.3 years. The most common causes of ESRD in the patients were hypertension in 30 (29.7%), Diabetes Mellitus in 12 (11.9%), Diabetes Mellitus with hypertension in 14 (13.9%); and unknown causes in 14 (13.9%). oral fungal infection (OFI) was found in 32% of the ESRD patients and 11% of the controls (p=0.007). An extensive OFI, defined as frequent fungal hyphae formations in oral mucosal smear layers, was found in 3% of the PD and 17% of the HD patients. Oral lesions, defined as clinical signs associated with OFI such as erythematous oral stomatitis, membranous candidiasis or angular cheilitis, were found in 37% of the patients with OFI, while 5% of the patients without findings of fungal infection presented oral lesions associated with OFI (p=0.0002). Furthermore, patients with self-reported mouth dryness were three times more likely (p=0.02) to be diagnosed with OFI. Mucosal disorders were observed in 24% of the patients. Furred tongue, scrotal tongue, and deficiency glossitis were seen in 8%, 6%, and 3% of the patients, respectively, and the frequency of herpes simplex and gingivitis were 3% and 2%, respectively.

Gábor L Kovács[76] stated that the patients who have Type 1 Diabetes with nephropathy and hypertension, 50% will go on to develop end-stage renal disease within a decade. 80% of people who have Type 1 Diabetes and microalbuminuria will progress to overt nephropathy (i.e. proteinuria characterized by > 300 mg albumin excreted daily), whereas only 20-40% of those with Type 2 Diabetes over a period of 15 years will progress.

Britt B Newsome et al[77] conducted a study on 87,094 patients, and found there were statistically significant interactions between creatinine change and race (*P*=.04), age (*P*<001), history of hypertension (*P*=.01), and history of Diabetes Mellitus (*P*<.001) with respect to the outcome of ESRD,

Ritz E, Bergis K, Strojek K and Keller C[78] found that Diabetic Nephropathy in patients with type II diabetes has become the most frequent cause of End stage Renal Failure in Germany. Preventive measures, i. e. near normal glycemia and particularly antihypertensive treatment, have been proven to interfere with progression of renal failure in diabetic nephropathy. Early recognition is possible by testing for urinary albumin (microalbuminuria). In patients with Diabetic Nephropathy, blood pressure should be lowered to values well within the range of normotension by dietary salt restriction and antihypertensive drug therapy

Barry i. Freedman et al[79] conducted a study in which the family histories were obtained from 4365 dialysis patients, 856 (20%) reported having a family history of ESRD

Udayakumar P et al[80] conducted a study on One hundred patients with CRF on hemodialysis and found Oral changes included macroglossia with teeth markings (35%), xerostomia (31%), ulcerative stomatitis (29%), angular cheilitis (12%) and uremic breath (8%). Some rare manifestations of CRF like uremic frost, gynecomastia and pseudo-Kaposi's sarcoma were also observed. Ulcerative stomatitis seen in 29% is reported to occur in patients with blood urea level more than 150mg/ml .

Shu-Fen Chuang et al[81] conducted a study in which they found that the incidence of uremic odour in Diabetic groups (27.9%). Uremic odour is associated with the accumulation of urea in the saliva. Higher incidence of uremic odour may correlate with higher urea in saliva of CRF patients. Incidence of mucosal petechia / ecchymosis was 20.9% in the diabetic. Patients with poor glycemic control also presented with a higher incidence of tongue/mucosa pain and tongue coating. Additionally, oral ulceration was rare. The mucosal petechia/ecchymosis is associated with the anticoagulants in hemodialysis sessions. Low incidence of these soft tissue lesions may reflect well controlled status of anticoagulants in these patients

P. Mosannen Mozaffari et al[82] stated that, one of the early symptoms may be a bad metallic taste and unpleasant odour in the mouth particularly in the morning. This uremic fetor, an ammoniacal odour is a typical sign of all uremic patients which is caused by the high concentration of urea in the saliva and its subsequent breakdown to ammonia. Salivary urea level correlates well with the BUN so that saliva can be used as a non invasive diagnostic tool. An acute rise in BUN (>150 mg/dl) may result in uremic stomatitis which disappear 2 to 3 weeks after medical intervention and decreasing BUN. Four of 300 patients with uremia were observed to have probable uremic stomatitis in the 1930s, while in 1964 another 4 affected patients were reported from a group of 262 patients with renal disease.

Uremic stomatitis is considered as a kind of chemical burn. The red burning mucosa is covered with gray exudates and would be ulcerative later. Four types of uremic stomatitis have been described: erythemopultaceous, ulcerative, hemorrhagic and hyperkeratotic. This lesion is painful and appears on the ventral surface of tongue and anterior mucosal surfaces. The most prevalent mucosal finding is pale mucosa as a result of normochromic/normocytic anaemia caused by decreasing of erythropoietin but increasing hemolysis due to dialysis procedure and uremic toxins. Lichenoid reaction (drug induced) and pyogenic granuloma are frequently observed in CRF patients. Caroten –like material deposition gives oral mucosa an orange-red color is seen. Gingival bleeding, petechia and ecchymosis develop in labial and buccal mucosa, soft palate and tongue borders as a result of qualitative and to a lesser degree, quantitative platelet defects. Anticoagulants used for hemodialysis can be a predisposing factor.

A M El Nahas et al[83] found that the ratio of plasma urea to creatinine concentration accurately reflected the dietary protein intake, it rose to 110 during the high protein diet and subsequently fell to 40 during the low protein diet (p <0 025).

Sanjay Kumar Agarwal et al[51], stated that A serum creatinine persistently >1.8mg% for 8–12 weeks in the absence of any reversible factor was the criterion to diagnose Chronic Renal Failure.

Agarwal et al[84] screened.4900 persons in urban communities of Delhi and found a .79% point prevalence of persons with serum creatinine more than1.8 mg/dL.

Robert N. Foley et al[85] found that the initial laboratory findings in patients starting renal replacement therapy (RRT) have also changed considerably from year to year. On comparing 1996 and 2005, initial hemoglobin levels rose from 9.3 to 10.2 g/dl, blood urea nitrogen fell from 94.0 to 83.3 mg/dl, serum creatinine fell from 8.5 to 6.8 mg/dl, and estimated GFR rose from 7.7 to 10.1 ml/min per 1.73 m^2.

De La Rosa García E et al[66] evaluated 229 individuals, Two adult groups were studied: Group A: ESRD DM on dialysis, and group B: non-ESRD DM (serum creatinine <2.0 mg/dl). Group A 99, and Group B 130 pts. Signs and symptoms prevalence was higher in group A: uremic breath (48.5%), unpleasant taste (45.5%) and xerostomia (44.4%) being the most frequent ones. Oral Lesions were also more prevalent in group A. The most frequent Oral Lesions were dry, fissured lips (28.3%), saburral tongue (18.2%) and candidiasis (17.2%). No difference was found in candidiasis prevalence between groups. Candidiasis was found associated to xerostomia and smooth tongue only in group A. The high prevalence of uremic fetor, xerostomia, saburral tongue and candidiasis in group A, could be tried as warning signs on the possibility of non diagnosed advanced renal disease in other diabetic patients

Sowell SB[86], Carl W and Wood RH[87], Hovinga J et al[88] found Uremic stomatitis is often a clinical finding in cases of advanced disease. There are two forms of this stomatitis; often, they correspond with an acute rise in BUN levels. The erythemopultaceous form is characterized by red, burning mucosa covered with a gray exudate and pseudomembrane; the ulcerative form is characterized by frank ulceration with redness and a pultaceous covering. The exact etiology of uremic stomatitis remains unknown, but it is suspected to be a chemical like burn or a loss of the tissue's resistance to normal and/or traumatic influences. These lesions are commonly painful and most often appear on the ventral tongue and anterior mucosal surfaces. These lesions usually heal spontaneously, with resolution of the underlying uremia and lowering of BUN levels.

Vesterinen M et al[89] conducted a cross-sectional study in the Helsinki University Central Hospital, Finland, on 148 patients with different kinds of kidney disease at predialysis state. Data from medical records, clinical oral examination, saliva, and mucosal yeast counts were analyzed and compared between the disease groups. Of the patients, 53 (36%) had Diabetic Nephropathy (29 patients with type 1, 24 patients with Type 2 Diabetes). Compared with other CKD patients, diabetic patients had poor glycemic control as expected (mean HbA1C) 8.0% vs 5.9%, ($p<0.01$). Diabetic patients also had more dental caries (mean number of carious teeth 5.1 vs 3.1, $p<0.01$) and lower salivary flow rates than other CKD patients (stimulated salivary flow 1.2 ml/min vs 1.6 ml/min, $p<0.05$). No difference

between groups was observed in periodontal health and yeast counts. Diabetic Nephropathy patients indeed had worse dental health in comparison to CKD group. Diabetic Nephropathy did not seem to affect periodontal health more severely than the other kidney diseases

Gavaldá et al[90] examined the oral mucosa of individuals with chronic renal failure and noted several mucosal lesions, uremic stomatitis and Candida infections in 37% of these patients.

Thorman R et al[75] evaluated 93 ESRD patients and 45 age- and gender-matched controls. In total, 34 patients were treated with peritoneal dialysis (PD) and 59 with hemodialysis (HD). OFI was found in 32% of the ESRD patients and 11% of the controls (p=0.007). An extensive oral fungal infection (OFI), defined as frequent fungal hyphae formations in oral mucosal smear layers, was found in 3% of the PD and 17% of the HD patients. Oral lesions, defined as clinical signs associated with OFI such as erythematous oral stomatitis, membranous candidiasis or angular cheilitis, were found in 37% of the patients with OFI, while 5% of the patients without findings of fungal infection presented oral lesions associated with OFI. Furthermore, patients with self-reported mouth dryness were three times more likely to be diagnosed with OFI.

De la Rosa-García E et al[91] performed a study in 90 patients of which Fifty (55.6%) men and forty (44.4%) women were studied. Sixty percent of the patients had at least one OL. Oral candidiasis (OC) was found in 18.7%; 13% had lesions clinically compatible with hairy leukoplakia

(CHL). An association was found between OC and CHL ($P<0.05$). Saburral tongue (ST) was found in 22% of the patients and gingival hyperplasia (GH) in 49%.

Safia A. Al-Attas et al[92] conducted a study on 150 Diabetics. They found that the number of patients with candidal carriage from the oral cavity was higher in patients with Type 1 Diabetes than in type 2 ($P=.003$).

E. de la Rosa García et al[93] conducted a study on ESRD DM and DM groups, in that order, consisting of 103 and 130 patients respectively. No differences were found in age (57.9 ± 11.4 *vs* 58.5 ± 11.5 years, p = 0.716), sex distribution, or schooling. Age at diagnosis of Type 2 Diabetes was 38.5 ± 14.0 *vs* 47.8 ± 12.1 years (p < 0.001). In ESRD DM patients, the median known duration of diabetes before dialysis was 17 years (1-39), and the median time on dialysis 7 months (1-88). Fourteen (13.6%) ESRD DM and 29 (22.3%) DM patients reported current or prior smoking (p = 0.088); 45.6% and 26.9% (p = 0.003) reported unpleasant taste, and dry mouth (p = 0.011).

REVIEW OF LITERATURE FOR DRUGS CAUSING ESRD AND NESRD IN DIABETES MELLITUS

Abe M et al[94] stated that Conventional oral hypoglycemic agents, such as sulfonylurea (SU), are not suitable due to the risk of prolonged hypoglycemia; furthermore, metformin is contraindicated for moderate to advanced CKD. Therefore, in order to achieve good glycemic control,

insulin injection therapy remains the mainstay of treatment in diabetic patients with moderate to advanced CKD, particularly in those receiving dialysis therapies. However, some agents have been used even in patients with CKD. Repaglinide and mitiglinide are rapid- and short-acting insulinotropic SU receptor ligands. They are rarely accompanied by hypoglycemia, and are attractive therapeutic options even in the dialysis population. In addition, alpha-glucosidase inhibitors are rarely accompanied by hypoglycemia and are administered without dose adjustments in dialysis patients. However, the National Kidney Foundation Kidney Disease Outcomes Quality Initiative guidelines recommended that alpha-glucosidase inhibitors should be avoided in patients with advanced stage CKD and on dialysis. Furthermore, mitiglinide is not currently used in the US. Thus, recommended oral antidiabetic agents differ between countries. Moreover, dipeptidyl peptidase-4 inhibitors and incretin mimetics are new antihyperglycemic agents, which may be used more frequently in the future in patients with Type 2 Diabetes and CKD. Here, we describe the pharmacokinetics, metabolism, clinical efficacy, and safety of oral Antidiabetic agents for patients with CKD, including those receiving dialysis

Jean-François Yale[95] found that in chronic renal failure, the oral agents that can be used therefore include the insulin secretagogues repaglinide and nateglinide and the thiazolidinediones (rosiglitazone and pioglitazone) with caution. Insulin also can be used safely in renal failure.

Jorge l Gross et al[74] stated that Metformin should not be used when serum creatinine is >1.5 mg/dl in men and >1.4 mg/dl in women due to the increased risk of lactic acidosis. Sulfonylureas and their metabolites, except glimepiride, are eliminated via renal excretion and should not be used in patients with decreased renal function Thus; most type 2 diabetic patients with Diabetic Nephropathy should be treated with insulin.

M. Greenwood et al[96] stated that Insulin given by injection may cause pain and swelling of the salivary glands. The oral hypoglycaemic metformin may produce a metallic taste. Sulphonylurea hypoglycaemics such as gliclazide and glibenclamide have been implicated in causing oral lichenoid eruptions, erythema multiforme and orofacial neuropathy such as burning tongue.

Janelle C Nisbet et al[97] found that life-threatening lactic acidosis can occur, caused by accumulation of metformin, and that risk factors for this include renal impairment, old age and doses over 2 g per day. The estimated prevalence of life-threatening lactic acidosis is one to five cases per 1,00,000 with mortality in reported cases up to 50%.

Devasmita Choudhury et al[98] suggested the Use of metformin and the first-generation sulfonylurea agents chlorpropamide, tolbutamide and tolazamide, as well as the α-glucosidase inhibitors acarbose and miglitol, should be avoided in patients with advanced CKD or ESRD, in light of their association with metabolic acidosis and prolonged hypoglycemia.

STUDY TOPIC

To assess oral signs, symptoms and oral lesions type and prevalence, in diabetic patients with End Stage Renal Disease (ESRD-DM) and compare them with analogous findings in Non-End Stage Renal Disease (NON-ESRDDM) group.

STUDY DESIGN

The present study is a comparative Study.

STUDY DURATION

This study was conducted between June 2010 to May 2011 in the department of Oral Medicine and Radiology of Ragas Dental College and Hospital, and Voluntary Health Services, Adyar, Chennai.

STUDY POPULATION

The study population includes patients reporting to Ragas Dental College & Hospital, Chennai, and Voluntary Health Services, Adyar, Chennai, who were diagnosed as Diabetic patients with End Stage Renal Disease (ESRD-DM) and with Non-End Stage Renal Disease (NESRD-DM) group.

Sample size – 200 Patients

1. 100 Diabetic patients with End Stage Renal Disease
2. 100 Diabetic patients with Non-End Stage Renal Disease

OBTAINING APPROVAL FROM THE AUTHORITIES

Permission from the ethical committee of the Ragas Dental College & Hospital, and Voluntary Health Services, Adyar, Chennai was obtained

before starting the study for interrogating and examining the patients. All patients participating in the study had to give written informed consent for participation in English or Tamil accordingly.

INSTRUMENTS USED

1. Dental chair with halogen lamp
2. Disposable latex gloves
3. Mouth mask
4. Plain mouth mirror
5. Dental probe
6. Mettalic scale
7. 2 X 2 gauze
8. Torch Light

METHODOLOGY

The 200 Diabetic patients, who are diagnosed as having Non-End Stage Renal Disease and End Stage Renal Disease undergoing dialysis will be taken up for the study. These patients will undergo general clinical examination to exclude HIV. Patients are examined for the presence of intra oral manifestations of Diabetic patients with End Stage Renal Disease (ESRD-DM) and with Non-End Stage Renal Disease (NESRD-DM) like saburral tongue, smooth tongue, burning tongue, candidiasis, dry and fissured lips, petechiae or ecchymoses, ulcerative or uremic stomatitis, herpes simplex, angular cheilitis, uremic fetor, xerostomia, pale mucosa.

END STAGE RENAL DISEASE IN DIABETIC PATIENTS

INCLUSION CRITERIA

1. Both sexes
2. Patient in age group of 12 years and above
3. Diabetic patients with End Stage Renal Disease on Dialysis

EXCLUSION CRITERIA

1. Patients with HIV infection

NON-END STAGE RENAL DISEASE IN DIABETIC PATIENTS

INCLUSION CRITERIA

1. Both sexes
2. Patient in age group of 12 years and above
3. Diabetic patients with Non-End Stage Renal Disease, Serum Creatinine is less than or equal to 2.0mg/dl

EXCLUSION CRITERIA

1. Patients with HIV infection
2. Patients with Serum Creatinine Level >2.0mg/dl

Data was collected from subjects fulfilling the above said criteria.

CLINICAL CRITERIA FOR DIAGNOSIS

SABURRAL TONGUE

Yellowish-white plaque on tongue dorsum, which could not be scraped-off by a blunt instrument. Slightly elongated filiform papillae and bacterial accumulation were found[66].

FISSURED TONGUE

It is characterized by grooves that vary in depth and are noted along the dorsal and lateral aspects of the tongue[99].

CANDIDIASIS

It is the most common fungal infection. It has various forms in which pseudo-membranous type has loosely attached membrane which when removed leaves a raw bleeding area; erythematous type is a successor to pseudo-membranous candidiasis which is red with diffuse borders and plaque-like or nodular candidiasis appears as a thick white plaque[100].

DRY AND FISSURED LIPS

The lips are dry, peeling or chapped. Breaks may appear on the surface, and the lips may become painful and may bleed[100].

PETECHIAE OR ECCHYMOSES

Purpura is the appearance of red or purple discolorations on the skin that do not blanch on applying pressure. They are caused by bleeding underneath the skin / Mucosa.

Purpura measure 0.3–1 cm (3–10 mm), whereas petechiae measure less than 3 mm, and ecchymoses greater than 1 cm.

Petechia refers to one of the three major classes of purpuric skin/mucus membrane conditions. Purpuric eruptions are classified by size into three broad categories. Petechiae are generally used to

refer to the smallest of the three classes of purpuric skin/mucous membrane eruptions, those that measure less than 3 mm.

Petechiae may be a sign of thrombocytopenia (low platelet counts) when platelet function is inhibited (e.g., as a side effect of medications or during certain infections), or in clotting factor deficiencies. They may also occur when excessive pressure is applied to tissue (e.g., when a tourniquet is applied to an extremity or with excessive coughing or vomiting).

By definition, ecchymoses are 1 to 2 cm in size or larger, and are therefore larger than petechiae (1–2 mm). A subcutaneous purpura larger than 1 centimeter[101]

SMOOTH TONGUE

Smooth tongue is a condition characterized by a smooth glossy tongue that is often tender /painful. The atrophy of papillae, resulting in a smooth tongue. The tongue may be pale or erythematous and may appear small or enlarged[102]

BURNING TONGUE

Burning Tongue is a condition characterized by a burning or tingling sensation on the lips, tongue, or entire mouth. This condition appears more often in women, specifically women after menopause, than men. Pain typically is low or nonexistent in the morning and builds up over the course of the day[102]

ULCERATIVE OR UREMIC STOMATITIS

An acute rise in BUN (>150 mg/dl) may result in uremic stomatitis which disappear 2 to 3 weeks after medical intervention and decreasing BUN.

Uremic stomatitis is considered as a kind of chemical burn. The red burning mucosa is covered with gray exudates and would be ulcerative later. Four types of uremic stomatitis have been described: erythemopultaceous, ulcerative, hemorrhagic and hyperkeratotic.

The erythemopultaceous form is characterized by red, burning mucosa covered with a gray exudates and pseudomembrane.

The ulcerative form is characterized by frank ulceration with redness and a pultaceous covering.

Clinically, adherent white lesions arise on the dorsal, ventral, and lateral parts of the tongue, as well as in the buccal, labial, or retro-molar areas.4 However, some patients may have an exudate from the oral mucosa, together with ulcerative lesions of the skin[82].

HERPES LABIALIS

The presence of clusters of vesicles which ruptures to form round, symmetric, shallow ulcers which coalesce to form larger ulcer with scalloped borders and marked surrounding erythema along the vermilion border of the lips[102].

ANGULAR CHEILITIS

Angular cheilitis is an inflammatory lesion at the labial commissure, or corner of the mouth, and often occurs bilaterally. The condition manifests as deep cracks or splits. In severe cases, the splits can bleed when the mouth is opened and shallow ulcers or a crust may form.

It is infected fissures of the commissures of the mouth which may ulcerate or develop super added candidal infection surrounded by erythema caused due to nutritional deficiencies or decreased vertical dimension of the complete denture[102]

UREMIC FETOR

Uremic fetor was identified when the patient had a urine-odor breath in persons with uremia[66]. The odor occurs from the smell of ammonia, which is created in the saliva as a breakdown product of urea. Uremic fetor is usually associated with an unpleasant metallic taste (dysgeusia)[81].

XEROSTOMIA

A diagnosis of xerostomia was made when a dry or sticky mucosa was found, and when the patient reported a dry mouth; saliva flow was not measured[66]

PALE MUCOSA

Pallor is condition caused by a reduced amount of oxyhaemoglobin in the skin or mucous membrane, a pale colour which

can be caused by illness, emotional shock or stress, stimulant use, lack of exposure to sunlight, anaemia or genetics.

It can develop suddenly or gradually, depending on the cause. It is not usually clinically significant unless it is accompanied by a general pallor (paleness of the lips, tongue, palms, mouth and other regions with mucous membranes)[102].

LICHEN PLANUS

Oral lichen planus is a mucocutaneous disease that affects skin, mucosa or both. It has both red and white components. It is usually bilateral with white striations called as Wickham's striae and commonly occurs on the buccal mucosa, tongue and gingiva. On palpation, the surface texture rough with loss of suppleness[102].

INVESTIGATIONS REQUIRED

Blood Sample Collection

Blood samples are taken from the vein in the antecubital fossa. The tourniquet is set around the upper arm of the subject, search for the proper vein by inspecting and palpating and then sterilize the injection site. The vein can be anchored by placing the thumb about two centimeters below the vein and pulling gently to make the skin a little taut. After that, the needle, beveled upward, should be pushed smoothly and quickly into the vein, to minimize the possibility of hemolysis as a result of vascular damage. Immediately after the insertion, the tourniquet should be released to

minimize the effect of hemoconcentration. 5 ml of venous blood was drawn and the serum was separated by centrifugation, supernatant was aspirated. The samples were centrifuged no later than 30 minutes after the sample was drawn. EDTA and Sodium Fluoride were added to prevent the coagulation of blood. All samples were centrifuged at 3000 rpm for 10 min to remove particulate materials. This freshly obtained serum was used immediately for biochemical analysis

Biochemical Analysis

Estimation of Creatinine in Serum

Modified Jaffe's Kinetic Method[103] was used to estimate the levels of Creatinine in serum.

Reagents

1. L1 : Picric Acid Reagent
2. L2 : Buffer Reagent
3. S : Creatinine Standard (2 mg/dl)

Procedure

100µL of the freshly obtained serum will be mixed with 0.1 mL of creatinine Standard (S), 0.5ml of picric acid reagent (L1) and buffer reagent (L2) each in test tube. Mix well and and the supernatant is read at 520 nm in a spectrophotometer (photometer 5010 V5+, ROBERT RIELE KG, BERLIN). The levels of creatinine in serum is expressed in term of mg/Dl

Estimation of Urea Level in Serum

Modified Berthelot Method[104] was used to estimate the levels of urea level in serum.

Reagents

1. L1 : COLOR Reagent
2. L2 : Enzyme Reagent
3. L3 : BASE Reagent

Procedure

10µL of the freshly obtained serum will be mixed with 0.5 ml of COLOR Reagent (L1), Add 0.5 ml of ENZYME Reagent (L2) mix gently, and incubate at 37°C for five minutes. Add 2.0 ml of BASE Reagent (L3), mix and incubate at 37°C for 5 minutes. Measure the absorbance of the mix at 630 nm by photometer (photometer 5010 V5+, ROBERT RIELE KG, BERLIN). The levels of Urea in serum is expressed in term of mg/dL

Estimation of Fasting Blood Glucose Level in Serum

GOD-POD Method[105] was used to estimate the levels of Fasting blood glucose in serum.

Reagents

1. L1 : Glucose standard Reagent

Procedure

10µL of the freshly obtained serum will be mixed with 1000µL of Glucose standard Reagent (L1) in the test tube. Mix well & incubate for 15 min at room temperature or 7 min at 37 ^{0}C. Measure the absorbance of the

mix at 505 nm by photometer (photometer 5010 V5+, ROBERT RIELE KG, BERLIN) at 630nm. The levels of Fasting blood glucose in serum is expressed in term of mg/Dl

Estimation of Hemoglobin Level in Blood

The Cyanmethemoglobin Method[106] was used to estimate the levels of hemoglobin in blood.

Reagents

1. L1 : cyanmethemoglobin reagent (Drabkin's solution)

Procedure

Pipette 5 ml of cyanmethemoglobin reagent into each tube. Add 20 µl of the blood sample into test tube. Allow tube to stand for 10 minutes. Read Absorbance (A) in the spectrophotometer at 540 nm, by photometer (photometer 5010 V5+, ROBERT RIELE KG, BERLIN). The levels of hemoglobin level in Blood is expressed in term of g/dL

STATISTICAL ANALYSIS

All the data were entered in Microsoft excel sheets. Statistical analysis was done using SPSS software SYSTAT version 7.0

Mean: defined as sum of values (X) divided by the number of values (N) and denoted by

P > 0.05 = Difference is not significant

$$\text{Mean}(X) = \frac{\sum X}{N}$$

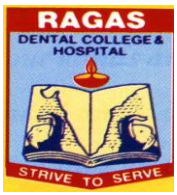
Chi Square Test

$$X^2 = \frac{\text{sum of (observed frequency – expected frequency)}^2}{\text{Expected frequency}} = \frac{\sum (O-E)^2}{E}$$

P ≤ 0.05 = Difference is significant (S)

P ≤ 0.01 = Difference is highly significant (S)

P ≤ 0.001 = Difference is very highly significant (HS)



RAGAS DENTAL COLLEGE & HOSPITAL

2/102, East Coast Road, Uthandi, Chennai - 600119

DEPARTMENT OF ORAL MEDICINE & RADIOLOGY

CASE SHEET PROFORMA

Date:

Name:

Age:

Sex:

Occupation:

Income:

Address:

Phone number:

Tobacco related habits:

Dietary habits:

Medical history:

1. Systemic disease:
2. Diabetes mellitus:

a) Duration of diabetes:

b) Family history:

c) Medication:

Investigations:

Serum creatinine level:

Blood glucose level: fasting sugar:

Serum urea level:

Oral lesion present:

- Saburral tongue :
- Candidiasis:
- Dry and fissured lips:
- Petechiae or ecchymoses:
- Smooth tongue:
- Burning tongue:
- Ulcerative or uremic stomatitis:
- Herpes simplex:
- Angular cheilitis:
- Uremic fetor:
- Xerostomia:
- Pale mucosa:
- Other lesions:

Figure 1: Armamentarium for Clinical Examination

Figure 2: Armamentarium for Blood Investigation

Figure 3: Aramamentarium for Estimation of Serum Creatinine, Blood Glucose Level, Blood Urea Level, Hemoglobin Level

Figure 4 :Laboratory Centrifuge Machine

Figure 5: Spectrophotometer for Estimation of Circulating Immune Complex

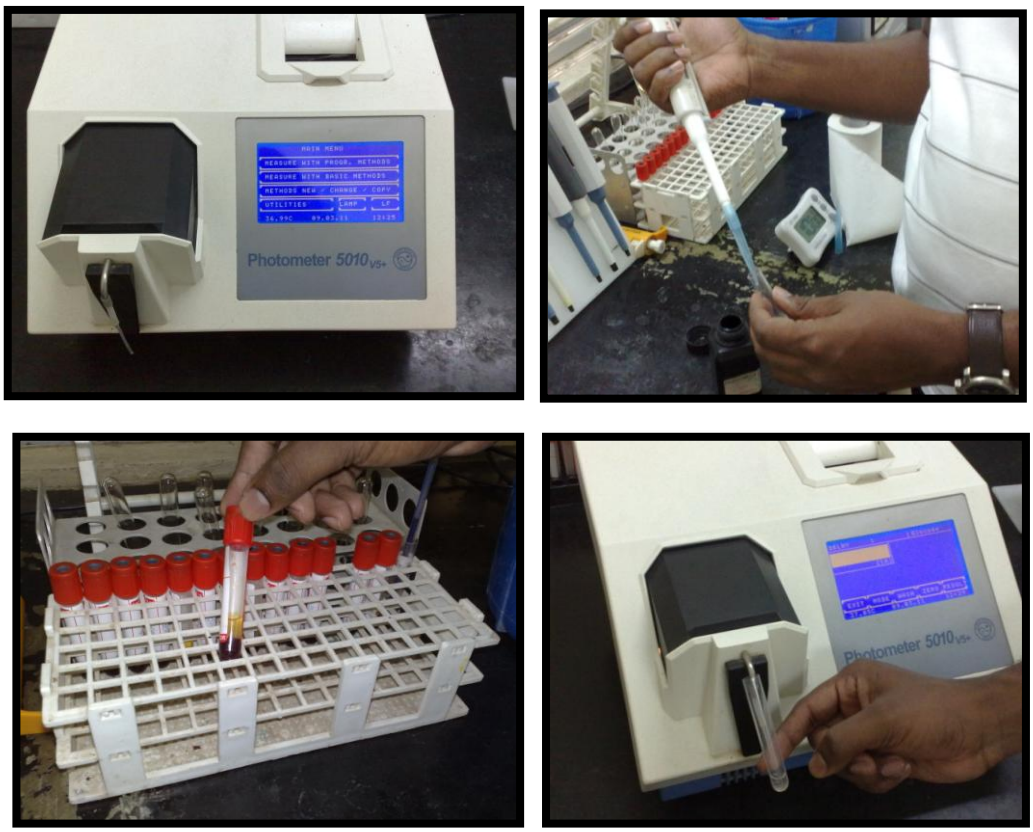


Figure 6 : Hemodialysis Procedure

Figure 7 : Saburral Tongue

Figure 8 : Fissured Tongue

Figure 9 : Oral Candidiasis

Figure 10 : Dry and Fissured Lips

Figure 11: Petechiae or Ecchymoses

Figure 12: Smooth Tongue

Figure 13: Ulcerative or Uremic Stomatitis

Figure 14: Herpes Simplex

Figure 15: Angular Cheilitis

Figure 16: Xerostomia

Figure 17: Pale Mucosa

Figure 18: Oral Lichen Planus

The present study is a Comparative study which was conducted in the Department of Oral Medicine and Radiology of Ragas Dental College and Hospital, Uthandi, Chennai and Voluntary Health Services, Adyar, Chennai. It was devised to compare the oral signs, symptoms and oral lesions type and prevalence in Diabetic patients with End Stage Renal Disease (ESRD-DM) and with Non-End Stage Renal Disease (NESRD-DM) group. The study was conducted between June 2010-March 2011 on a total of 200 diabetic patients, who are diagnosed as having End Stage Renal Disease-undergoing dialysis and Non-End Stage Renal Disease. The data obtained from the study were statistically analyzed. The results extracted are compared with various variables included in the study and are presented here.

Table 1: Distribution of ESRD and NESRD among Diabetes Mellitus patients with relation to Sex

The study group consists of 200 subjects, 79 patients were male and 121 patients were female. Out of the 100 ESRD patients, 41 patients (51.90%) are found to be male and 59 patients (48.80%) are found to be female .Among the 100 NESRD patients, 38 patients (48.10%) are male and 62 patients (51.20%) were female.

The sex wise distribution of subjects were found to be **statistically non significant**, which means that both ESRD and NESRD among Diabetes Mellitus patients were not similar with respect to sex in distribution with **P value is ≥ 0.664**

TABLE 2: Distribution of ESRD and NESRD among Diabetes Mellitus patients with relation to age

The age of the subjects included in the study ranges between 1-100 years. So the subjects were divided into five age groups which are as follows: 12-40 years, 41-60 years, 61-80 years and 81-100 years. Among 200 patients, 72 were in age group of 12-40 years, 101 patients were in age group of 41-60 years, 24 patients were in age group of 61-80 years, and 3 patients were in the age group of 81-100 years. Among the 100 patients in ESRD- diabetes mellitus patients, 22 (30.56%) were between 12-40yrs, 59 (58.41%) were between 40- 60 yrs, 17 (70.83%) were between 60-80 yrs, 2 (66.67%) were between 80-100 yrs, the mean age group affected by ESRD is 50.77. Among the 100 patients in NESRD- diabetes mellitus patients, 50 (69.44%) were between 12- 40 yrs, 42 (41.59%) were between 40-60 yrs, 7 (29.17%) were between 60-80 yrs, 1 (33.33%) were between 80-100 yrs, the mean age group affected by NESRD is 40.82.

The age wise distribution of subjects were found to be **statistically significant**, which means that there exists correlation among the 2 groups with respect to age in distribution with P **value ≤ 0.000**.

TABLE 3: Distribution of ESRD and NESRD among Diabetes Mellitus patients with relation to habits.

Among the 200 patients, 141 patients did not have any habits, 35 patients have smoking habits, and 24 patients have tobacco related habits. Among 100 ESRD patients, 60 (42.60%) patients did not have any habits,

22 (62.90%) patients have smoking habits, and 18 (75%) patients have tobacco related habits. Among 100 NESRD patients, 81 (57.40%) patients did not have any habits, 13 (37.10%) patients have smoking habits, and 6 (25%) patients have tobacco related habits

The distribution of subjects based on habits were found to be **statistically significant**, which means that there exists correlation among the 2 groups with respect to habit in distribution with **P value ≤ 0.003**

TABLE 4: Distribution of ESRD and NESRD among Diabetes Mellitus patients with relation to diet

Among the 200 patients, 106 patients were on renal diet, 91 patients were on vegetarian diet and 3 patients were on Non vegetarian diet. Among 100 ESRD patients, 29 patients (27.4%) were on renal diet, 68 patients (74.70%) were on vegetarian diet and 3 (100%) patients were on Non vegetarian diet. Among 100 NESRD patients, 77 patients (72.6%) were on renal diet, 23 patients (25.30%) were on vegetarian diet and 0 (0%) patients were on Non vegetarian diet

The distribution of subjects based on diet were found to be **statistically significant**, which means that there exists correlation among the 2 groups with respect to habit in distribution with **P value ≤ 0.000**

TABLE 5: Distribution of ESRD and NESRD among Diabetes Mellitus patients with relation to Type of Diabetes Mellitus

Among the 200 patients, 39 patients were IDDM (Insulin–Dependent Diabetes Mellitus) and 161 patients were NIDDM (Non Insulin –

Dependent Diabetes Mellitus). Among 100 ESRD patients, 17 (43.6%) were IDDM (Insulin – Dependent Diabetes Mellitus) patients and 83 (51.60%) were NIDDM (Non Insulin – Dependant Diabetes Mellitus) patients. Among 100 NESRD patients, 22 (56.4%) were IDDM (Insulin – Dependant Diabetes Mellitus) patients and 78 (48.4%) were NIDDM (Non Insulin – Dependant Diabetes Mellitus) patients

The distribution of subjects based on Type of Diabetes Mellitus were found to be **statistically not significant**, which means that there exists no correlation among the 2 groups with respect to Type of Diabetes Mellitus in distribution with **P value ≥ 0.372**

TABLE 6: Distribution of ESRD and NESRD among Diabetes Mellitus patients with relation to Duration of Diabetes Mellitus

The Duration of Diabetes Mellitus of the subjects included in the study ranges between 1yr - 80 years. So the subjects were divided into four groups which are as follows: 1-12 yrs, 13-40 years, 41-60 years, and 61-80 years. Among the 200 patients, 140 were between 1-12 yrs, 59 were between 13-40yrs, 1 (100%) were between 41- 60 yrs, 0 (0%) were between 61-80 yrs. Among the 100 patients in ESRD- diabetes mellitus patients, 54 (38.58%) were between 1-12 yrs, 45 (76.28%) were between 13-40yrs, 1 (100%) were between 41- 60 yrs, 0 (0%) were between 61-80 yrs. The mean duration of Diabetes Mellitus age group affected by ESRD is 14.39yrs. Among the 100 patients in NESRD- Diabetes Mellitus patients, 86 (61.42%) were between 1-12 yrs, 14 (23.72%) were between 13-40 yrs, 0 (0%) were

between 41-60 yrs, 0 (0%) were between 61-80 yrs, the mean duration of Diabetes Mellitus group affected by NESRD is 5.82yrs.

The distribution of subjects based on duration of Diabetes Mellitus was found to be **statistically very highly significant**, which means that there exists correlation among the 2 groups with respect to age in distribution with **P value ≤ 0.000**.

TABLE 7: Distribution of ESRD and NESRD among Diabetes Mellitus patients with relation to family history of Diabetes Mellitus

Among the 200 patients, 46 patients had family history of Diabetes Mellitus and 154 patients did not have any family history of Diabetes Mellitus. Among 100 ESRD patients, 24 (52.2%) had family history of Diabetes Mellitus and 76 (49.40%) patients did not have any family history of Diabetes Mellitus. Among 100 NESRD patients, 22 (47.8%) had family history of Diabetes Mellitus and 78 (50.60%) patients did not have any family history of Diabetes Mellitus

The distribution of subjects based on family history of Diabetes Mellitus were found to be **statistically not significant**, which means that there exists no correlation among the 2 groups with respect to family history of Diabetes Mellitus in distribution with **P value ≥ 0.737**

TABLE 8: Distribution of ESRD and NESRD among Diabetes Mellitus patients with relation to fasting blood sugar level

The fasting blood sugar level of the subjects included in the study ranges between 0-200 mg/dl. So the subjects were divided into three groups

which are as follows: less than 120 mg/dl, 120-200 mg/dl, and more than 200 mg/dl. Among the 200 patients, 71 were found to be less than 120 mg/dl, 79 were between 120-200 mg/dl, 50 were more than 200 mg/dl. Among the 100 patients in ESRD- Diabetes Mellitus patients, 30 (42.25%) were found to be less than 120 mg/dl, 42 (53.17%) were between 120-200 mg/dl, 28 (56%) were more than 200 mg/dl. the mean value for the fasting blood sugar level in ESRD patients is 161.63mg/dl. Among the 100 patients in NESRD- diabetes mellitus patients, 41 (57.75%) were found to be less than 120 mg/dl, 37 (46.83%) were between 120-200 mg/dl, 22 (44%) were more than 200 mg/dl. The mean value for the fasting blood sugar level in NESRD patients is 153.18mg/dl

The distribution of subjects based on fasting blood sugar level were found to be **statistically not significant**, which means that there exists no correlation among the 2 groups with respect to fasting blood sugar level in distribution with **P value $\geq$ 0.330**

TABLE 9: Distribution of ESRD and NESRD among Diabetes Mellitus patients with relation to Hypertension

Among the 200 patients, 40 patients were hypertensive and 160 patients were normotensive. Among 100 ESRD patients, 26 (65%) were hypertensive patients and 74 (46.30%) were normotensive. Among 100 NESRD patients, 14 (35%) were hypertensive patients and 86 (53.70%) were normotensive.

The distribution of subjects based on Hypertension were found to be **statistically significant**, which means that there exists correlation among the 2 groups with respect to habit in distribution with **P value ≥ 0.034**

TABLE 10: Distribution of ESRD and NESRD among Diabetes Mellitus patients with relation to hemoglobin level

The hemoglobin level of the subjects included in the study ranges between 0-20 mg/dl. So the subjects were divided into three groups which are as follows: less than 10 mg/dl, 10-15 mg/dl, more than 15 mg/dl. Among the 200 patients 63 were found to be less than 10 mg/dl, 128 were between 10-15 mg/dl, 9 were more than 15 mg/dl.Among the 100 patients in ESRD- diabetes mellitus patients, 56 (88.89%) were found to be less than 10 mg/dl, 41 (32.03%) were between 10-15 mg/dl, 3 (33.33%) were more than 15 mg/dl. the mean value for the hemoglobin level in ESRD patients is 9.81mg/dl. Among the 100 patients in NESRD- diabetes mellitus patients, 7 (11.11%) were found to be less than 10 mg/dl, 87 (67.97%) were between 10-15 mg/dl, 6 (66.67%) were more than 15 mg/dl. The mean value for the hemoglobin level in NESRD patients is 12.3mg/dl

The distribution of subjects based on Concentration of hemoglobin level were found to be **statistically significant**, which means that there exists correlation among the 2 groups with respect to Concentration of hemoglobin level in distribution with **P value ≤ 0.000**

TABLE 11: Distribution of ESRD and NESRD among Diabetes Mellitus patients with relation to serum creatinine

The Serum creatinine of the subjects included in the study ranges between 0-10 mg/dl. So the subjects were divided into three groups which are as follows: less than 2 mg/dl, 2-10 mg/dl, more than 10 mg/dl. Among the 200 patients, 100 were found to be less than 2 mg/dl, 91 were between 2-10 mg/dl, 9 were more than 10 mg/dl. Among the 100 patients in ESRD-Diabetes Mellitus patients, 0 (0%) were found to be less than 2 mg/dl, 91 (100%) were between 2-10 mg/dl, 9 (100%) were more than 10 mg/dl. The mean value for the Serum creatinine in ESRD patients is 4.334mg/dl. Among the 100 patients in NESRD- Diabetes Mellitus patients, 100 (100%) were found to be less than 2 mg/dl, (0%) were between 2-10 mg/dl, 0 (0%) were more than 10 mg/dl. the mean value for the Serum creatinine in NESRD patients is 0.945mg/dl.

The distribution of subjects based on Concentration of Serum creatinine were found to be **statistically significant**, which means that there exists correlation among the 2 groups with respect to Concentration of Serum creatinine in distribution with **P value ≤ 0.000**.

TABLE 12: Distribution of ESRD and NESRD among Diabetes Mellitus patients with relation to blood urea level

The blood urea level of the subjects included in the study ranges between 0-150 mg/dl. So the subjects were divided into three groups which are as follows: less than 50 mg/dl, 50-100 mg/dl, more than 100 mg/dl. Among the 200 patients, 137 were found to be less than 50 mg/dl, 40 were between 50-100 mg/dl, 23 (100%) were more than 100 mg/dl. Among the

100 patients in ESRD- diabetes mellitus patients, 37 (27%) were found to be less than 50 mg/dl, 40 (100%) were between 50-100 mg/dl, 23 (100%) were more than 100 mg/dl. the mean value for the blood urea level in ESRD patients is 73.72mg/dl. Among the 100 patients in NESRD- Diabetes Mellitus patients, 100 (73%) were found to be less than 50 mg/dl, 0 (0%) were between 50-100 mg/dl, 0 (0%) were more than 100 mg/dl. the mean value for the blood urea level in ESRD patients is 28.66mg/dl.

The distribution of subjects based on Concentration of blood urea level were found to be **statistically significant**, which means that there exists correlation among the 2 groups with respect to Concentration of blood urea level in distribution with **P value ≤ 0.000**

TABLE 13: Distribution of ESRD and NESRD among Diabetes Mellitus patients with relation to medication for Diabetes Mellitus

The study group consists of 200 subjects, 58 patients had taken metformin, 95 patients had taken sulphonyl urea and 47 patients had taken insulin. Out of the 100 ESRD patients, 52 patients (89.70%) had taken metformin, 40 (42.10%) patients had taken sulphonyl urea and 8 (17%) patients had taken insulin. Among 100 NESRD patients, 6 patients (10.30%) had taken metformin, 55 (57.90%) patients had taken sulphonyl urea and 39 (83%) patients had taken insulin.

The distribution of subjects based on medication for Diabetes Mellitus were found to be **statistically significant**, which means that there

exists correlation among the 2 groups with respect to medication for Diabetes Mellitus in distribution with **P value ≤ 0.000**

TABLE 14: Distribution of ESRD and NESRD among Diabetes Mellitus patients with relation to Saburral Tongue

Among the 200 patients, 24 patients had Saburral Tongue and 176 patients did not have Saburral Tongue. Among 100 ESRD patients, 19 (79.20%) had Saburral Tongue and 81 (46.00%) patients did not have Saburral Tongue. Among 100 NESRD patients, 5 (20.80%) had Saburral Tongue and 95 (54.00%) patients did not have Saburral Tongue.

The distribution of subjects based on presence of Saburral Tongue were found to be **statistically significant**, which means that there exists correlation among the 2 groups with respect to presence of Saburral Tongue in distribution with **P value ≤ 0.002**

TABLE 15: Distribution of ESRD and NESRD among Diabetes Mellitus patients with relation to Fissured Tongue

Among the 200 patients, 4 patients had Fissured Tongue and 196 patients did not have Fissured Tongue. Among 100 ESRD patients, 2 (50%) had Fissured Tongue and 98 (50%) patients did not have Fissured Tongue. Among 100 NESRD patients, 2 (50%) had Fissured Tongue and 98 (50%) patients did not have Fissured Tongue.

The distribution of subjects based on Fissured Tongue were found to be **statistically not significant**, which means that there exists no correlation

among the 2 groups with respect to Fissured Tongue in distribution with **P value ≤ 1.000**

TABLE 16: Distribution of ESRD and NESRD among Diabetes Mellitus patients with relation to Smooth Tongue

Among the 200 patients, 18 patients had smooth Tongue and 182 patients did not have smooth Tongue. Among 100 ESRD patients, 11 (61.10%) had smooth Tongue and 89 (48.90%) patients did not have smooth Tongue. Among 100 NESRD patients, 7 (38.90%) had smooth Tongue and 93 (51.10%) patients did not have smooth Tongue.

The distribution of subjects based on smooth Tongue were found to be **statistically not significant**, which means that there exists no correlation among the 2 groups with respect to smooth Tongue in distribution with **P value ≥ 0.323**

TABLE 17: Distribution of ESRD and NESRD among Diabetes Mellitus patients with relation to Burning Tongue

Among the 200 patients, 23 patients had Burning Tongue and 177 patients did not have Burning Tongue. Among 100 ESRD patients, 13 (56.50%) had Burning Tongue and 87 (49.20%) patients did not have Burning Tongue. Among 100 NESRD patients, 10 (43.50%) had Burning Tongue and 90 (50.80%) patients did not have Burning Tongue.

The distribution of subjects based on Burning Tongue were found to be **statistically not significant**, which means that there exists no correlation

among the 2 groups with respect to Burning Tongue in distribution with **P value ≥ 0.506**

TABLE 18: Distribution of ESRD and NESRD among Diabetes Mellitus patients with relation to Candidiasis

Among the 200 patients, 51 patients had Candidiasis and 149 patients did not have Candidiasis. Among 100 ESRD patients, 23 (45.10%) had Candidiasis and 77 (51.70%) patients did not have Candidiasis. Among 100 NESRD patients, 28 (54.90%) had Candidiasis and 72 (48.30%) patients did not have Candidiasis.

The distribution of subjects based on Candidiasis were found to be **statistically not significant**, which means that there exists no correlation among the 2 groups with respect to Candidiasis in distribution with **P value ≥ 0.417**

TABLE 19: Distribution of ESRD and NESRD among Diabetes Mellitus patients with relation to Dry and Fissured Lips

Among the 200 patients, 44 patients had Dry and Fissured Lips and 156 patients did not have Dry and Fissured Lips. Among 100 ESRD patients, 22 (50.00%) had Dry and Fissured Lips and 78 (50.00%) patients did not have Dry and Fissured Lips. Among 100 NESRD patients, 22 (50.00%) had Dry and Fissured Lips and 78 (50.00%) patients did not have Dry and Fissured Lips.

The distribution of subjects based on Dry and Fissured Lips were found to be **statistically not significant**, which means that there exists no

correlation among the 2 groups with respect to Dry and Fissured Lips in distribution with **P value ≥ 1.000**

TABLE 20: Distribution of ESRD and NESRD among Diabetes Mellitus patients with relation to Petechiae / Ecchymoses

Among the 200 patients, 20 patients had Petechiae / Ecchymoses and 180 patients did not have Petechiae / Ecchymoses. Among 100 ESRD patients, 18 (90.00%) had Petechiae / Ecchymoses and 82 (45.60%) patients did not have Petechiae / Ecchymoses. Among 100 NESRD patients, 2 (10.00%) had Petechiae / Ecchymoses and 98 (54.40%) patients did not have Petechiae / Ecchymoses.

The distribution of subjects based on presence of Petechiae / Ecchymoses were found to be **statistically significant**, which means that there exists correlation among the 2 groups with respect to presence of Petechiae / Ecchymoses in distribution with **P value ≤ 0.000**

TABLE 21: Distribution of ESRD and NESRD among Diabetes Mellitus patients with relation to Angular Chelitis

Among the 200 patients, 13 patients had Angular Chelitis and 187 patients did not have Angular Chelitis. Among 100 ESRD patients, 4 (30.80%) had Angular Chelitis and 96 (51.30%) patients did not have Angular Chelitis. Among 100 NESRD patients, 9 (69.20%) had Angular Chelitis and 91 (48.70%) patients did not have Angular Chelitis.

The distribution of subjects based on Angular Chelitis were found to be **statistically not significant**, which means that there exists no correlation

among the 2 groups with respect to Angular Chelitis in distribution with **P value ≥ 0.152**

TABLE 22: Distribution of ESRD and NESRD among Diabetes Mellitus patients with relation to Uremic Stomatitis

Among the 200 patients, 1 patient had Uremic Stomatitis and 199 patients did not have Uremic Stomatitis. Among 100 ESRD patients, 1 (100%) had Uremic Stomatitis and 99 (49.70%) patients did not have Uremic Stomatitis. Among 100 NESRD patients, 0 (0%) had Uremic Stomatitis and 100 (50.30%) patients did not have Uremic Stomatitis.

The distribution of subjects based on Uremic Stomatitis were found to be **statistically not significant**, which means that there exists no correlation among the 2 groups with respect to Uremic Stomatitis in distribution with **P value ≥ 0.316**

TABLE 23: Distribution of ESRD and NESRD among Diabetes Mellitus patients with relation to Uremic Fetor

Among the 200 patients, 53 patients had Uremic Fetor and 147 patients did not have Uremic Fetor. Among 100 ESRD patients, 52 (98.10%) had Uremic Fetor and 48 (32.70%) patients did not have Uremic Fetor. Among 100 NESRD patients, 1 (1.90%) had Uremic Fetor and 99 (67.30%) patients did not have Uremic Fetor.

The distribution of subjects based on presence of Uremic Fetor were found to be **statistically significant**, which means that there exists

correlation among the 2 groups with respect to presence of Uremic Fetor in distribution with **P value ≤ 0.000**

TABLE 24: Distribution of ESRD and NESRD among Diabetes Mellitus patients with relation to Xerostomia

Among the 200 patients, 82 patients had Xerostomia and 118 patients did not have Xerostomia. Among 100 ESRD patients, 42 (51.20%) had Xerostomia and 58 (49.20%) patients did not have Xerostomia. Among 100 NESRD patients, 40 (48.80%) had Xerostomia and 60 (50.80%) patients did not have Xerostomia.

The distribution of subjects based on Xerostomia were found to be **statistically not significant**, which means that there exists no correlation among the 2 groups with respect to Xerostomia in distribution with **P value ≥ 0.774**

TABLE 25: Distribution of ESRD and NESRD among Diabetes Mellitus patients with relation to Herpes Labialis

Among the 200 patients, 8 patients had Herpes Labialis and 192 patients did not have Herpes Labialis. Among 100 ESRD patients, 5 (62.50%) had Herpes Labialis and 95 (49.50%) patients did not have Herpes Labialis. Among 100 NESRD patients, 3 (37.50%) had Herpes Labialis and 97 (50.50%) patients did not have Herpes Labialis.

The distribution of subjects based on Herpes Labialis were found to be **statistically not significant**, which means that there exists no correlation

among the 2 groups with respect to Herpes Labialis in distribution with **P value ≥ 0.470**

TABLE 26: Distribution of ESRD and NESRD among Diabetes Mellitus patients with relation to Aphthous Ulcer

Among the 200 patients, 5 patients had Aphthous Ulcer and 195 patients did not have Aphthous Ulcer. Among 100 ESRD patients, 1 (20%) had Aphthous Ulcer and 99 (50.80%) patients did not have Aphthous Ulcer. Among 100 NESRD patients, 4 (80%) had Aphthous Ulcer and 96 (49.20%) patients did not have Aphthous Ulcer.

The distribution of subjects based on Aphthous Ulcer were found to be **statistically not significant**, which means that there exists no correlation among the 2 groups with respect to Aphthous Ulcer in distribution with **P value ≥ 0.174**

TABLE 27: Distribution of ESRD and NESRD among Diabetes Mellitus patients with relation to Pale Mucosa

Among the 200 patients, 59 patients had Pale Mucosa and 141 patients did not have Pale Mucosa. Among 100 ESRD patients, 45 (76.30%) had Pale Mucosa and 55 (39.00%) patients did not have Pale Mucosa. Among 100 NESRD patients, 14 (23.70%) had Pale Mucosa and 86 (61.00%) patients did not have Pale Mucosa.

The distribution of subjects based on presence of Pale Mucosa were found to be **statistically significant**, which means that there exists

correlation among the 2 groups with respect to presence of Pale Mucosa in distribution with **P value ≤ 0.000**

TABLE 28: Distribution of ESRD and NESRD among Diabetes Mellitus patients with relation to Unpleasant Taste

Among the 200 patients, 86 patients had Unpleasant Taste and 114 patients did not have Unpleasant Taste. Among 100 ESRD patients, 46 (53.50%) had Unpleasant Taste and 54 (47.40%) patients did not have Unpleasant Taste. Among 100 NESRD patients, 40 (46.50%) had Unpleasant Taste and 60 (52.60%) patients did not have Unpleasant Taste.

The distribution of subjects based on Unpleasant Taste were found to be **statistically not significant**, which means that there exists no correlation among the 2 groups with respect to Unpleasant Taste in distribution with **P value ≥ 0.391**

TABLE 29: Distribution of ESRD and NESRD among Diabetes Mellitus patients with relation to Stomatitis Medicamentosa

Among the 200 patients, 4 patients had Stomatitis Medicamentosa and 196 patients did not have Stomatitis Medicamentosa. Among 100 ESRD patients, 4 (100%) had Stomatitis Medicamentosa and 96 (49.00%) patients did not have Stomatitis Medicamentosa. Among 100 NESRD patients, 0 (0%) had Stomatitis Medicamentosa and 100 (51.00%) patients did not have Stomatitis Medicamentosa .

The distribution of subjects based on presence of Stomatitis Medicamentosa was found to be **statistically significant**, which means that

there exists correlation among the 2 groups with respect to presence of Stomatitis Medicamentosa in distribution with **P value ≤ 0.043.**

TABLE 30: Distribution of ESRD and NESRD among Diabetes Mellitus patients with relation to Lichen Planus

Among the 200 patients, 11 patients had Lichen Planus and 189 patients did not have Lichen Planus. Among 100 ESRD patients, 7 (63.60%) had Lichen Planus and 93 (49.20%) patients did not have Lichen Planus. Among 100 NESRD patients, 4 (36.40%) had Lichen Planus and 96 (50.80%) patients did not have Lichen Planus.

The distribution of subjects based on Lichen Planus were found to be **statistically not significant**, which means that there exists no correlation among the 2 groups with respect to Lichen Planus in distribution with **P value ≥ 0.352**

Table 1: Distribution of ESRD and NESRD among Diabetes Mellitus patients with relation to Sex

Group	Male		Female		Total number of patients	P-value
	Number	Percentage	Number	Percentage		
ESRD	41	51.90%	59	48.80%	100	≥ 0.664
NESRD	38	48.10%	62	51.20%	100	
Total	79	100%	121	100%	200	

P- Value is ≥ 0.664, this is statistically not significant

Table 2: Distribution of ESRD and NESRD among Diabetes Mellitus patients with relation to age

Group	Age								Total number of patients	Mean (yrs)	P-value
	12-40 yrs		41-60 yrs		61-80 yrs		81-100 yrs				
	No	%	No	%	No	%	No	%			
ESRD	22	30.56%	59	58.41%	17	70.83%	2	66.67%	100	50.77	≤ 0.000
NESRD	50	69.44%	42	41.59%	7	29.17%	1	33.33%	100	40.82	
total	72	100%	101	100%	24	100%	3	100%	200		

P- Value is ≤ 0.000; this is statistically very highly significant

Table 3: Distribution of ESRD and NESRD among Diabetes Mellitus patients with relation to habits

Group	No habits		Smoking		Tobacco		Total number of patients	P-value
	Number	Percentage	Number	Percentage	Number	Percentage		
ESRD	60	42.60%	22	62.90%	18	75.00%	100	≤ 0.003
NESRD	81	57.40%	13	37.10%	6	25.00%	100	
Total	141	100%	35	100%	24	100%	200	

P- Value is ≤ 0.003; this is statistically very highly significant

Table 4: Distribution of ESRD and NESRD among Diabetes Mellitus patients with relation to diet

Group	Renial Diet		Vegetarian		Non-Vegetarian		Total number of patients	P-value
	Number	Percentage	Number	Percentage	Number	Percentage		
ESRD	29	27.40%	68	74.70%	3	100.00%	100	≤ 0.000
NESRD	77	72.60%	23	25.30%	0	0.00%	100	
Total	106	100%	91	100%	3	100%	200	

P- Value is ≤ 0.000; this is statistically very highly significant

Table 5: Distribution of ESRD and NESRD among Diabetes Mellitus patients with relation to Type of Diabetes Mellitus

Group	IDDM		NIDDM		Total number of patients	P-value
	Number	Percentage	Number	Percentage		
ESRD	17	43.60%	83	51.60%	100	≥ 0.372
NESRD	22	56.40%	78	48.40%	100	
Total	39	100%	161	100%	200	

P- Value is ≥ 0.372, this is statistically not significant

Table 6: Distribution of ESRD and NESRD among Diabetes Mellitus patients with relation to Duration of Diabetes Mellitus

	Duration of Diabetes mellitus								Total number of patients	Mean (yrs)	P-value
	1-12yrs		13-40yrs		41-60 yrs		61-80 yrs				
	No	%	No	%	No	%	No	%			
ESRD	54	38.58%	45	76.28%	1	100%	0	0%	100	14.39	≤ 0.000
NESRD	86	61.42%	14	23.72%	0	0%	0	0%	100	5.82	
Total	140	100%	59	100%	1	100%	0	0%	200		

P- Value is ≤ 0.000; this is statistically very highly significant

Table 7: Distribution of ESRD and NESRD among Diabetes Mellitus patients with relation to family history of Diabetes Mellitus

Group	Family history of Diabetic Mellitus				Total number of patients	P-value
	Present		Absent			
	Number	Percentage	Number	Percentage		
ESRD	24	52.20%	76	49.40%	100	≥ 0.737
NESRD	22	47.80%	78	50.60%	100	
Total	46	100%	154	100%	200	

P- Value is ≥ 0.737, this is statistically not significant

Table 8: Distribution of ESRD and NESRD among Diabetes Mellitus patients with relation to fasting blood sugar level

Group	Fasting blood sugar level						Total number of patients	Mean	P-value
	<120 mg/dl		120 to 200 mg/dl		> 200 mg/dl				
	No	%	No	%	No	%			
ESRD	30	42.25%	42	53.17%	28	56%	100	161.63	≥ 0.330
NESRD	41	57.75%	37	46.83%	22	44%	100	153.18	
Total	71	100%	79	100%	50	100%	200		

P- Value is ≥ 0.330, this is statistically not significant

Table 9: Distribution of ESRD and NESRD among Diabetes Mellitus patients with relation to Hypertension

Group	Hypertension				Total number of patients	P-value
	Present		Absent			
	Number	Percentage	Number	Percentage		
ESRD	26	65.00%	74	46.30%	100	≤ 0.034
NESRD	14	35.00%	86	53.70%	100	
Total	40	100%	160	100%	200	

P- Value is ≤ 0.034, this is statistically significant

Table 10: Distribution of ESRD and NESRD among Diabetes Mellitus patients with relation to Hemoglobin level

Group	Hemoglobin (Hb) level						Total number of patients	Mean (mg/dl)	P-value
	<10 mg/dl		10 to 15 mg/dl		> 15 mg/dl				
	No	%	No	%	No	%			
ESRD	56	88.89%	41	32.03%	3	33.33%	100	9.81	≤ 0.000
NESRD	7	11.11%	87	67.97%	6	66.67%	100	12.3	
Total	63	100%	128	100%	9	100%	200		

P- Value is ≤ 0.000; this is statistically very highly significant

Table 11: Distribution of ESRD and NESRD among Diabetes Mellitus patients with relation to Serum Creatinine

	Serum creatinine						Total number of patients	Mean (mg/dl)	P-value
	<2 mg/dl		2 to 10 mg/dl		>10 mg/dl				
	No	%	No	%	No	%			
ESRD	0	0	91	100%	9	100%	100	4.334	≤ 0.000
NESRD	100	100%	0	0	0	0%	100	0.945	
Total	100	100%	91	100%	9	100%	200		

P- Value is ≤ 0.000; this is statistically very highly significant

Table 12: Distribution of ESRD and NESRD among Diabetes Mellitus patients with relation to Blood Urea Level

	Blood Urea Level						Total number of patients	Mean (mg/dl)	P-value
	<50 mg/dl		50 to 100 mg/dl		> 100 mg/dl				
	No	%	No	%	No	%			
ESRD	37	27%	40	100%	23	100%	100	73.72	≤ 0.000
NESRD	100	73%	0	0	0	0	100	28.66	
Total	137	100%	40	100%	23	100%	200		

P- Value is ≤ 0.000; this is statistically very highly significant

Table 13: Distribution of ESRD and NESRD among Diabetes Mellitus patients with relation to medication for Diabetes Mellitus

Group	Metformin		Sulphonylurea		Insulin		Total	P-value
	Number	Percentage	Number	Percentage	Number	Percentage		
ESRD	52	89.70%	40	42.10%	8	17.00%	100	≤ 0.000
NESRD	6	10.30%	55	57.90%	39	83.00%	100	
Total	58	100%	95	100%	47	100%	200	

P- Value is ≤ 0.000; this is statistically very highly significant

Table 14: Distribution of ESRD and NESRD among Diabetes Mellitus patients with relation to Saburral Tongue

Group	Saburral tongue				Total number of patients	P-value
	Present		Absent			
	Number	Percentage	Number	Percentage		
ESRD	19	79.20%	81	46.00%	100	≤ 0.002
NESRD	5	20.80%	95	54.00%	100	
Total	24	100%	176	100%	200	

P- Value is ≤ 0.002; this is statistically very highly significant

Table 15: Distribution of ESRD and NESRD among Diabetes Mellitus patients with relation to Fissured Tongue

Group	Fissured Tongue				Total number of patients	P-value
	Present		Absent			
	Number	Percentage	Number	Percentage		
ESRD	2	50.00%	98	50.00%	100	≥ 1.000
NESRD	2	50.00%	98	50.00%	100	
Total	4	100%	196	100%	200	

P- Value is ≥ 1.000, this is statistically not significant

Table 16: Distribution of ESRD and NESRD among Diabetes Mellitus patients with relation to Smooth Tongue

Group	Smooth Tongue				Total number of patients	P-value
	Present		Absent			
	Number	Percentage	Number	Percentage		
ESRD	11	61.10%	89	48.90%	100	≥ 0.323
NESRD	7	38.90%	93	51.10%	100	
Total	18	100%	182	100%	200	

P- Value is ≥ 0.323, this is statistically not significant

Table 17: Distribution of ESRD and NESRD among Diabetes Mellitus patients with relation to Burning Tongue

Group	Burning Tongue				Total number of patients	P-value
	Present		Absent			
	Number	Percentage	Number	Percentage		
ESRD	13	56.50%	87	49.20%	100	≥0.506
NESRD	10	43.50%	90	50.80%	100	
Total	23	100%	177	100%	200	

P- Value is ≥ 0.506, this is statistically not significant

Table 18: Distribution of ESRD and NESRD among Diabetes Mellitus patients with relation to Candidiasis

Group	Candidiasis				Total number of patients	P-value
	Present		Absent			
	Number	Percentage	Number	Percentage		
ESRD	23	45.10%	77	51.70%	100	≥ 0.417
NESRD	28	54.90%	72	48.30%	100	
Total	51	100%	149	100%	200	

P- Value is ≥ 0.417, this is statistically not significant

Table 19: Distribution of ESRD and NESRD among Diabetes Mellitus patients with relation to Dry and Fissured Lips

Group	Dry and Fissured Lips				Total number of patients	P-value
	Present		Absent			
	Number	Percentage	Number	Percentage		
ESRD	22	50.00%	78	50.00%	100	1.000
NESRD	22	50.00%	78	50.00%	100	
Total	44	100%	156	100%	200	

P- Value is ≥ 1.000, this is statistically not significant

Table 20: Distribution of ESRD and NESRD among Diabetes Mellitus patients with relation to Petechiae / Ecchymoses

Group	Petechiae / Ecchymoses				Total number of patients	P-value
	Present		Absent			
	Number	Percentage	Number	Percentage		
ESRD	18	90.00%	82	45.60%	100	≤ 0.000
NESRD	2	10.00%	98	54.40%	100	
Total	20	100%	180	100%	200	

P- Value is ≤ 0.000, this is statistically very highly significant

Table 21: Distribution of ESRD and NESRD among Diabetes Mellitus patients with relation to Angular Chelitis

Group	Angular Chelitis				Total number of patients	P-value
	Present		Absent			
	Number	Percentage	Number	Percentage		
ESRD	4	30.80%	96	51.30%	100	≥0.152
NESRD	9	69.20%	91	48.70%	100	
Total	13	100%	187	100%	200	

P- Value is ≥ 0.152, this is statistically not significant

Table 22: Distribution of ESRD and NESRD among Diabetes Mellitus patients with relation to Uremic Stomatitis

Group	Uremic Stomatitis				Total number of patients	P-value
	Present		Absent			
	Number	Percentage	Number	Percentage		
ESRD	1	100.00%	99	49.70%	100	≥0.316
NESRD	0	0.00%	100	50.30%	100	
Total	1	100%	199	100%	200	

P- Value is ≥ 0.316, this is statistically not significant

Table 23: Distribution of ESRD and NESRD among Diabetes Mellitus patients with relation to Uremic Fetor

Group	Uremic Fetor				Total number of patients	P-value
	Present		Absent			
	Number	Percentage	Number	Percentage		
ESRD	52	98.10%	48	32.70%	100	≤ 0.000
NESRD	1	1.90%	99	67.30%	100	
Total	53	100%	147	100%	200	

P- Value is ≤ 0.000; this is statistically very highly significant

Table 24: Distribution of ESRD and NESRD among Diabetes Mellitus patients with relation to Xerostomia

Group	Xerostomia				Total number of patients	P-value
	Present		Absent			
	Number	Percentage	Number	Percentage		
ESRD	42	51.20%	58	49.20%	100	≥ 0.774
NESRD	40	48.80%	60	50.80%	100	
Total	82	100%	118	100%	200	

P- Value is ≥ 0.774, this is statistically not significant

Table 25: Distribution of ESRD and NESRD among Diabetes Mellitus patients with relation to Herpes Labialis

Group	Herpes Labialis				Total number of patients	P-value
	Present		Absent			
	Number	Percentage	Number	Percentage		
ESRD	5	62.50%	95	49.50%	100	≥ 0.470
NESRD	3	37.50%	97	50.50%	100	
Total	8	100%	192	100%	200	

P- Value is ≥ 0.470, this is statistically not significant

Table 26: Distribution of ESRD and NESRD among Diabetes Mellitus patients with relation to Aphthous Ulcer

Group	Aphthous Ulcer				Total number of patients	P-value
	Present		Absent			
	Number	Percentage	Number	Percentage		
ESRD	1	20.00%	99	50.80%	100	≥ 0.174
NESRD	4	80.00%	96	49.20%	100	
Total	5	100%	195	100%	200	

P- Value is ≥ 0.174, this is statistically not significant

Table 27: Distribution of ESRD and NESRD among Diabetes Mellitus patients with relation to Pale Mucosa

Group	Pale Mucosa				Total number of patients	P-value
	Present		Absent			
	Number	Percentage	Number	Percentage		
ESRD	45	76.30%	55	39.00%	100	≤ 0.000
NESRD	14	23.70%	86	61.00%	100	
Total	59	100%	141	100%	200	

P- Value is ≤ 0.000; this is statistically very highly significant

TABLE 28: Distribution of ESRD and NESRD among Diabetes Mellitus patients with relation to Unpleasant Taste

Group	Unpleasant Taste				Total number of patients	P-value
	Present		Absent			
	Number	Percentage	Number	Percentage		
ESRD	46	53.50%	54	47.40%	100	≥0.391
NESRD	40	46.50%	60	52.60%	100	
Total	86	100%	114	100%	200	

P- Value is ≥ 0.391, this is statistically not significant

Table 29: Distribution of ESRD and NESRD among Diabetes Mellitus patients with relation to Stomatitis Medicamentosa

Group	Stomatitis Medicamentosa				Total number of patients	P-value
	Present		Absent			
	Number	Percentage	Number	Percentage		
ESRD	4	100.00%	96	49.00%	100	≤ 0.043
NESRD	0	0.00%	100	51.00%	100	
Total	4	100%	196	100%	200	

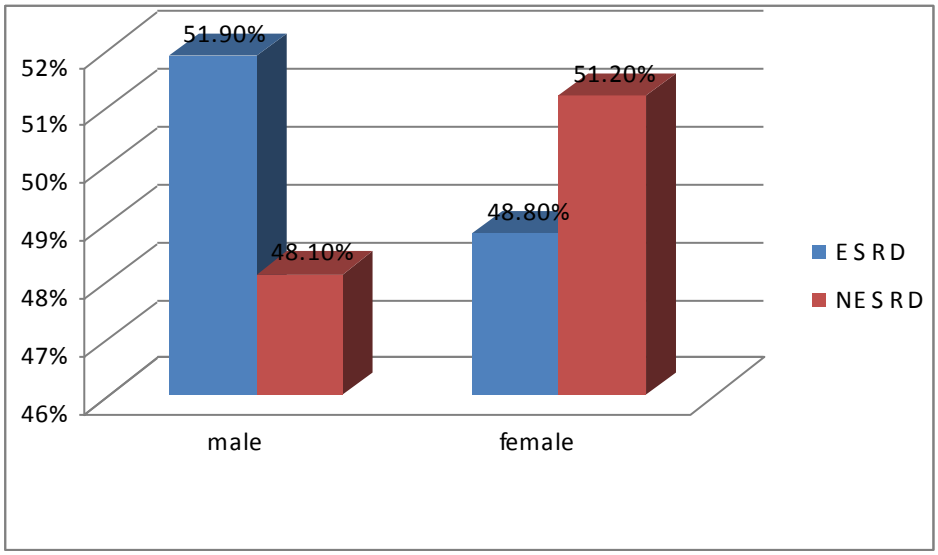
P- Value is ≤ 0.043, this is statistically significant

Table 30: Distribution of ESRD and NESRD among Diabetes Mellitus patients with relation to Lichen Planus

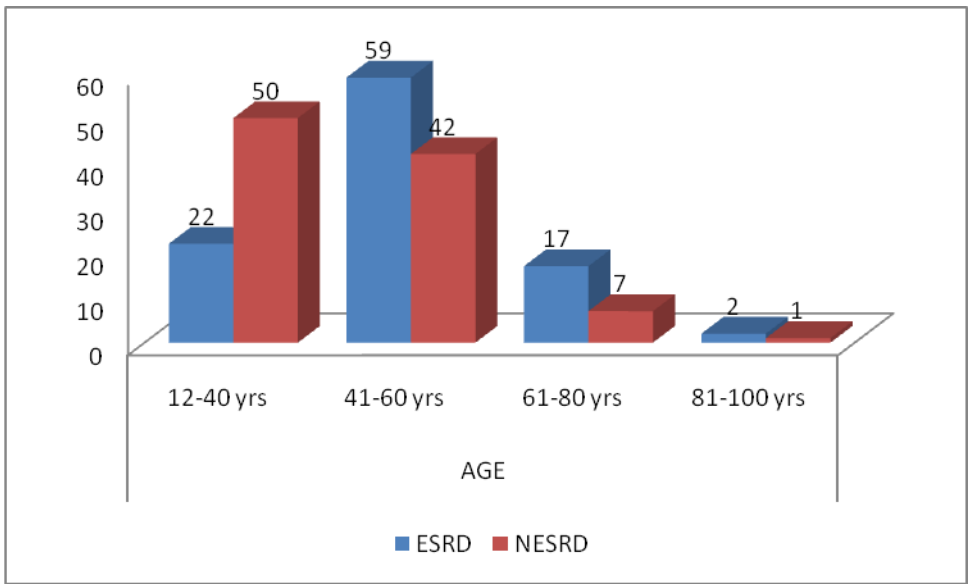
Group	Lichen Planus				Total number of patients	P-value
	Present		Absent			
	Number	Percentage	Number	Percentage		
ESRD	7	63.60%	93	49.20%	100	≥ 0.352
NESRD	4	36.40%	96	50.80%	100	
Total	11	100%	189	100%	200	

P- Value is ≥ 0.352, this is statistically not significant

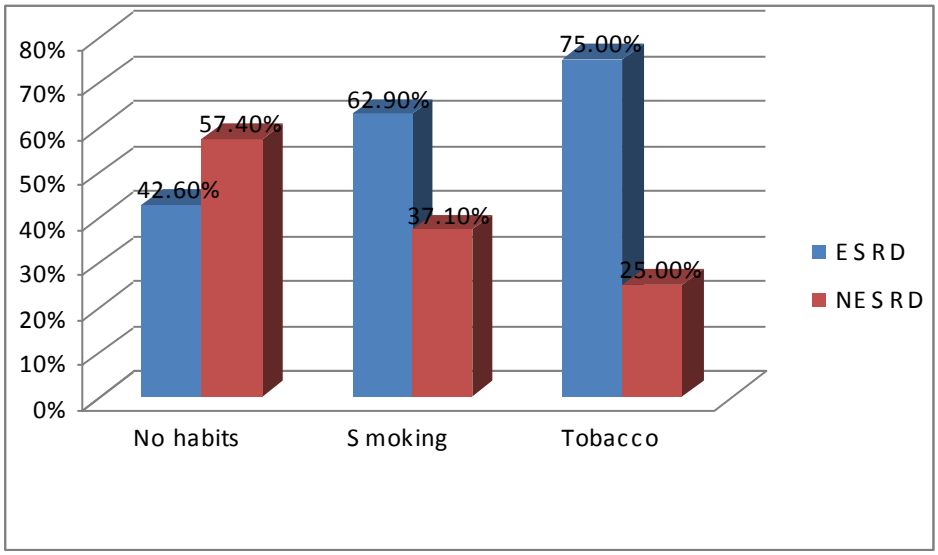
Graph 1: Distribution of ESRD and NESRD among Diabetes Mellitus patients with relation to Sex



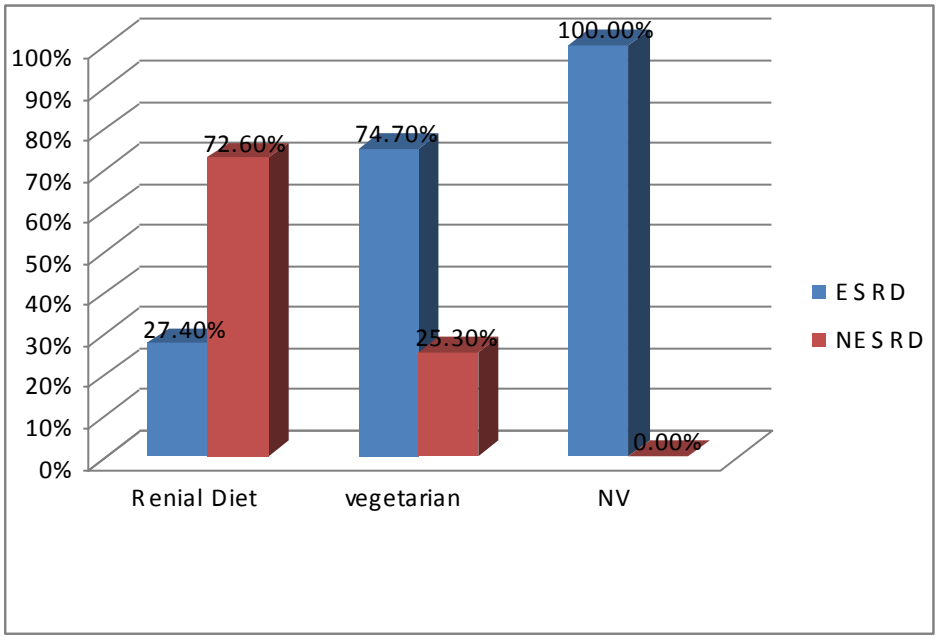
Graph 2: Distribution of ESRD and NESRD among Diabetes Mellitus patients with relation to Age



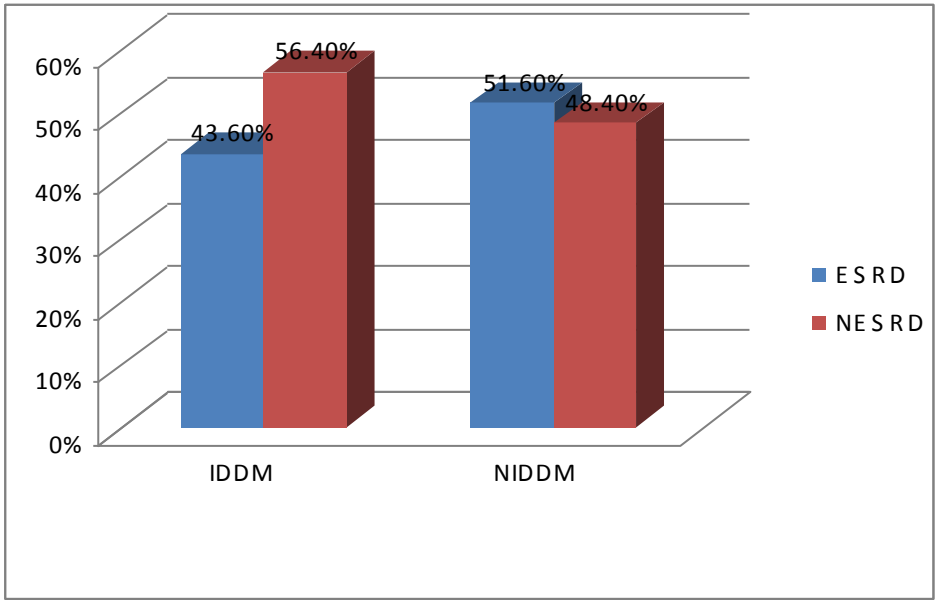
Graph 3: Distribution of ESRD and NESRD among Diabetes Mellitus patients with relation to Habits



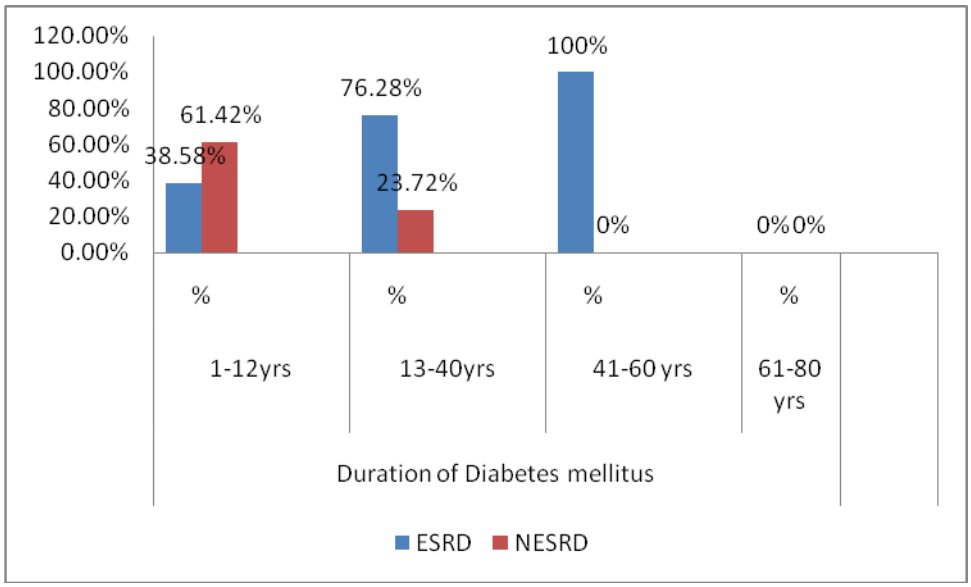
Graph 4: Distribution of ESRD and NESRD among Diabetes Mellitus patients with relation to Diet



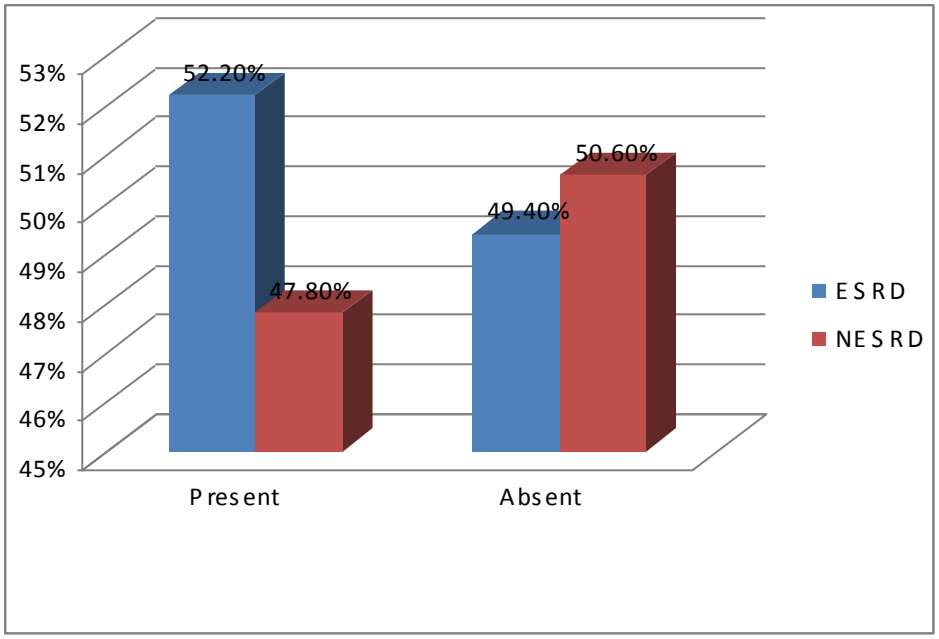
Graph 5: Distribution of ESRD and NESRD among Diabetes Mellitus patients with relation to Type of Diabetes Mellitus



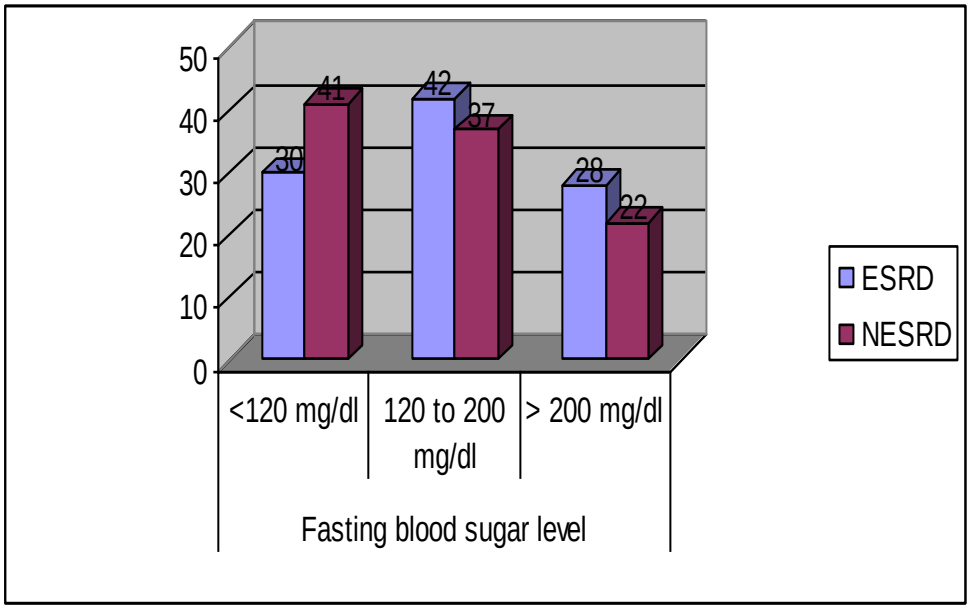
Graph 6: Distribution of ESRD and NESRD among Diabetes Mellitus patients with relation to Duration of Diabetes Mellitus



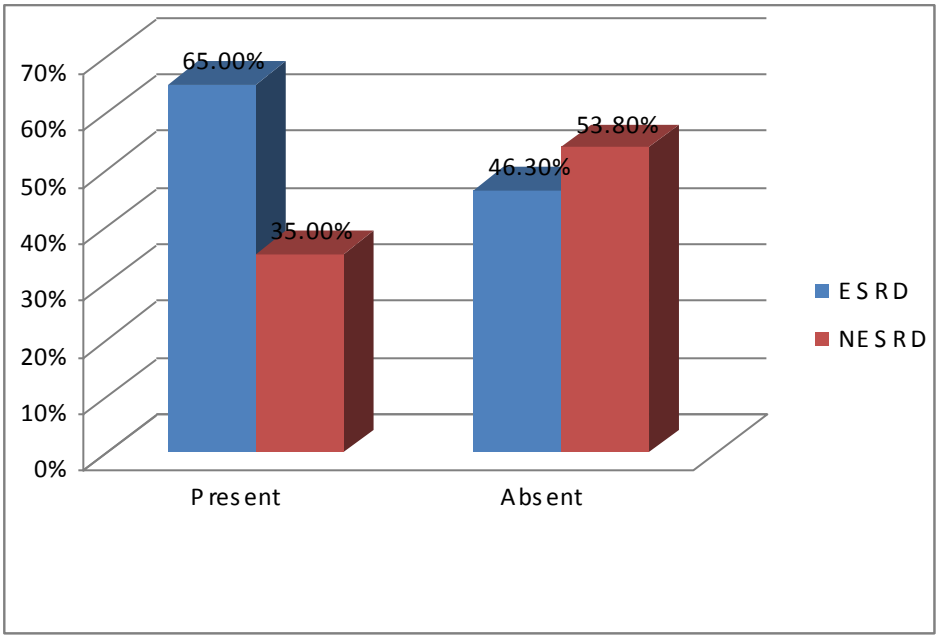
Graph 7: Distribution of ESRD and NESRD among Diabetes Mellitus patients with relation to family history of Diabetes Mellitus



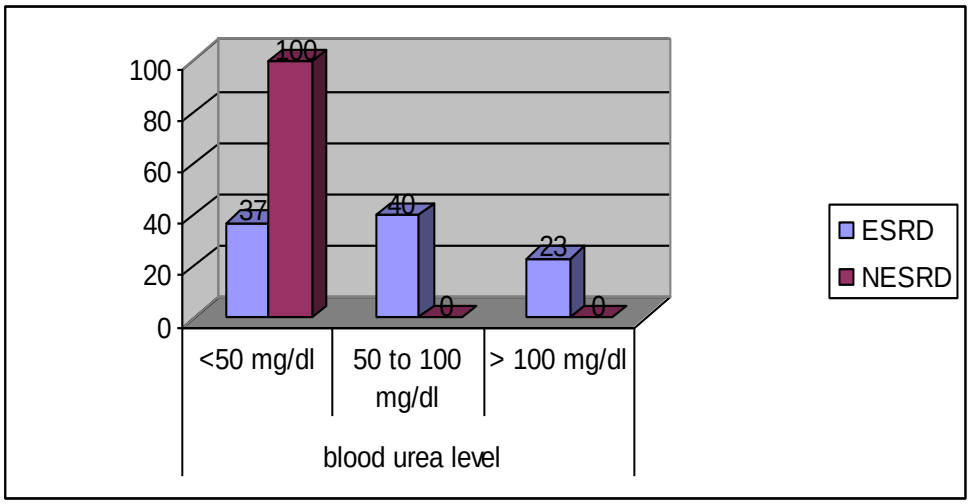
Graph 8: Distribution of ESRD and NESRD among Diabetes Mellitus patients with relation to Fasting Blood Sugar Level



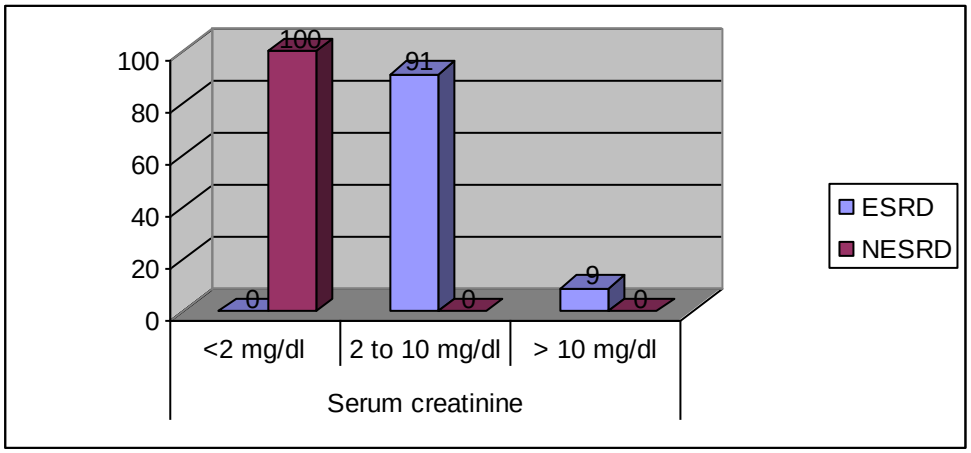
Graph 9: Distribution of ESRD and NESRD among Diabetes Mellitus patients with relation to Hypertension



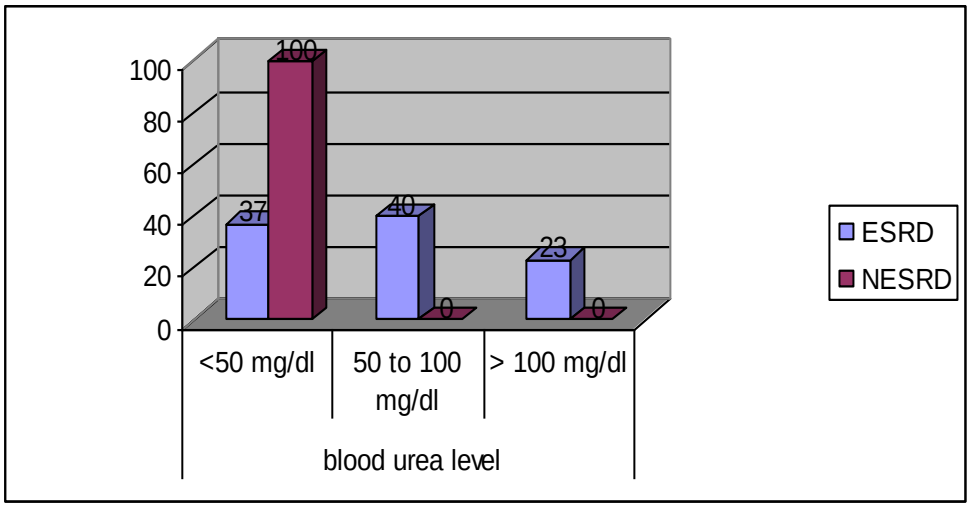
Graph 10: Distribution of ESRD and NESRD among Diabetes Mellitus patients with relation to Hemoglobin Level



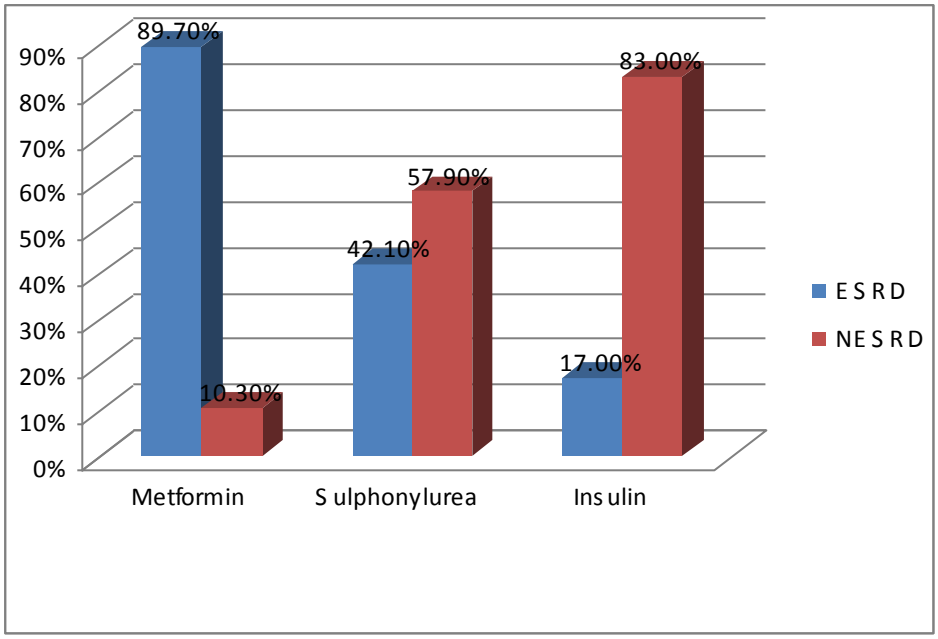
Graph 11: Distribution of ESRD and NESRD among Diabetes Mellitus patients with relation to Serum Creatinine



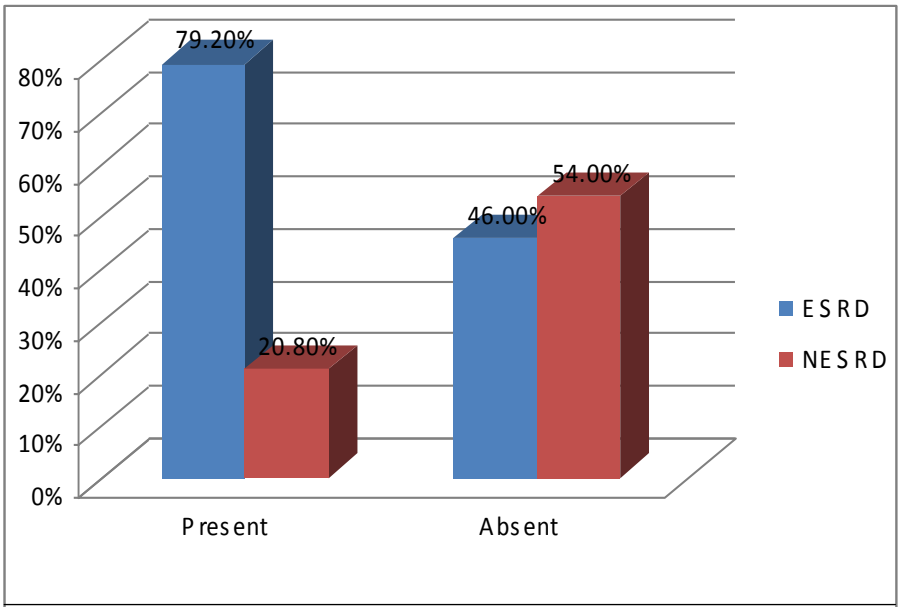
Graph 12: Distribution of ESRD and NESRD among Diabetes Mellitus patients with relation to Blood Urea Level



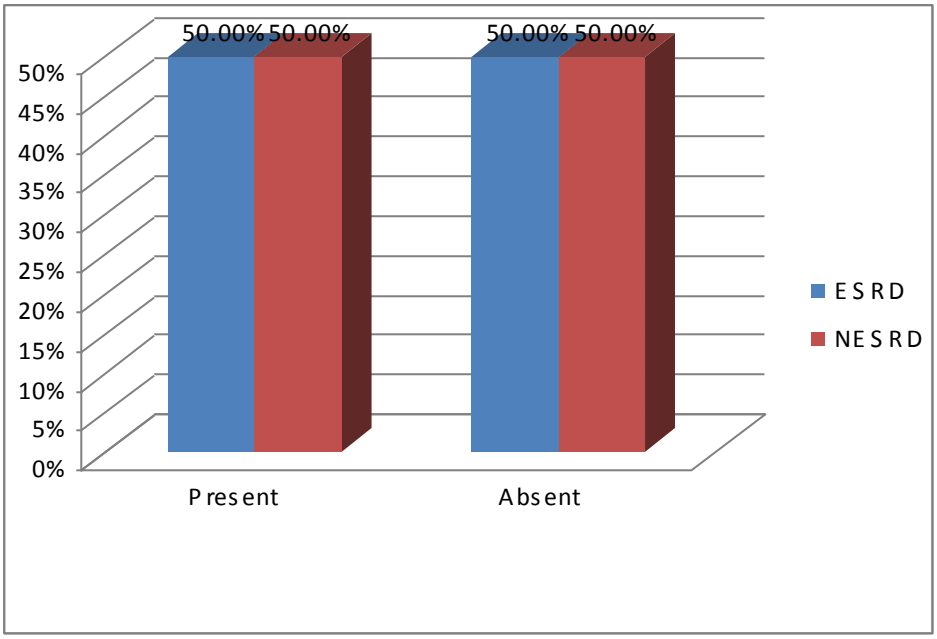
Graph 13: Distribution of ESRD and NESRD among Diabetes Mellitus patients with relation to medication for Diabetes Mellitus



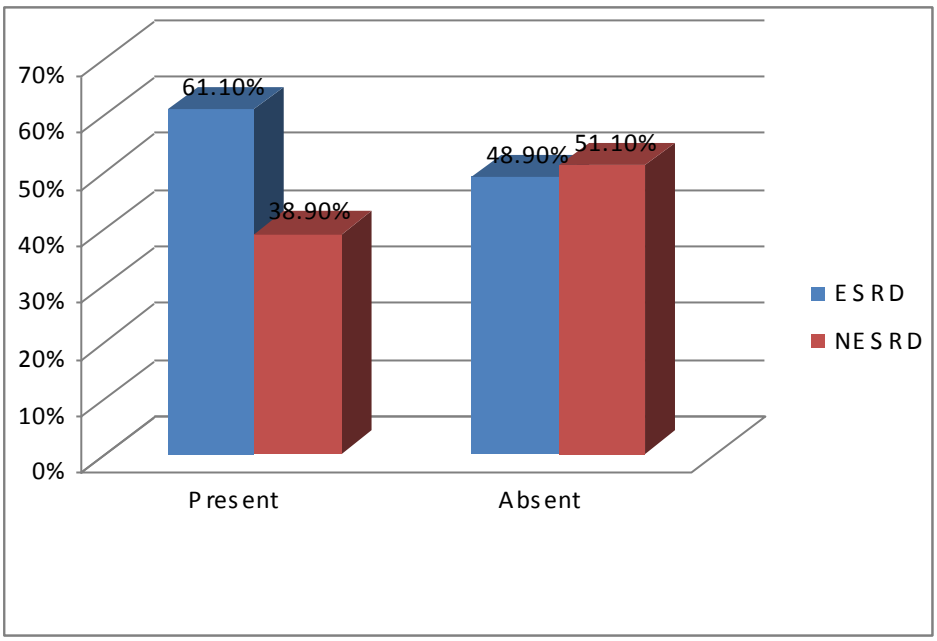
Graph 14: Distribution of ESRD and NESRD among Diabetes Mellitus patients with relation to Saburral Tongue



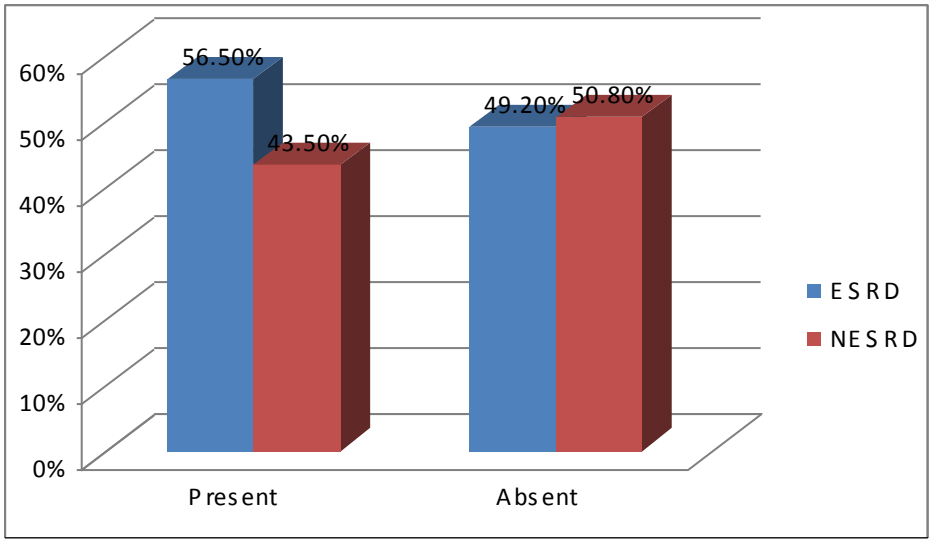
Graph 15: Distribution of ESRD and NESRD among Diabetes Mellitus patients with relation to Fissured Tongue



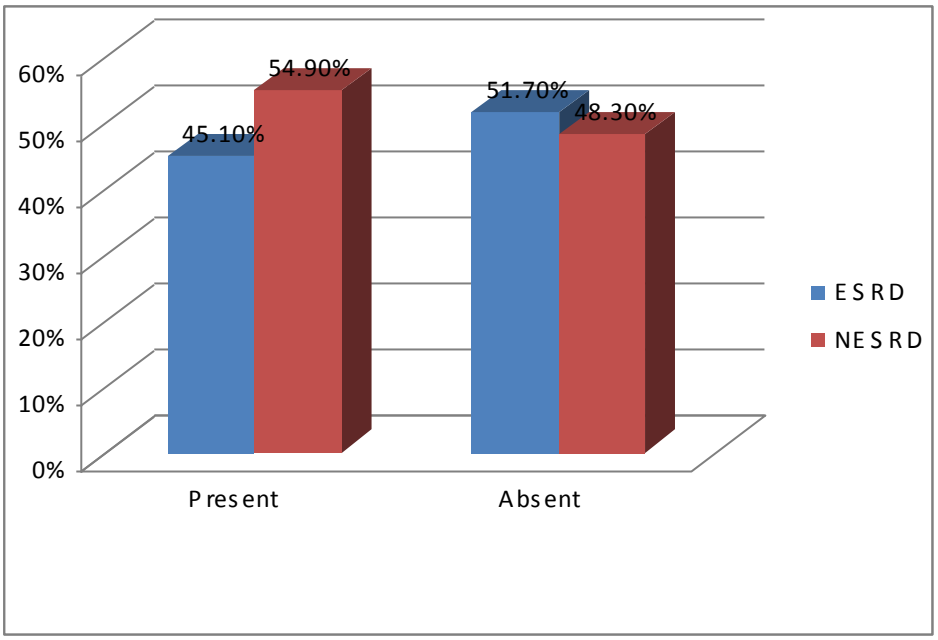
Graph 16: Distribution of ESRD and NESRD among Diabetes Mellitus patients with relation to Smooth Tongue



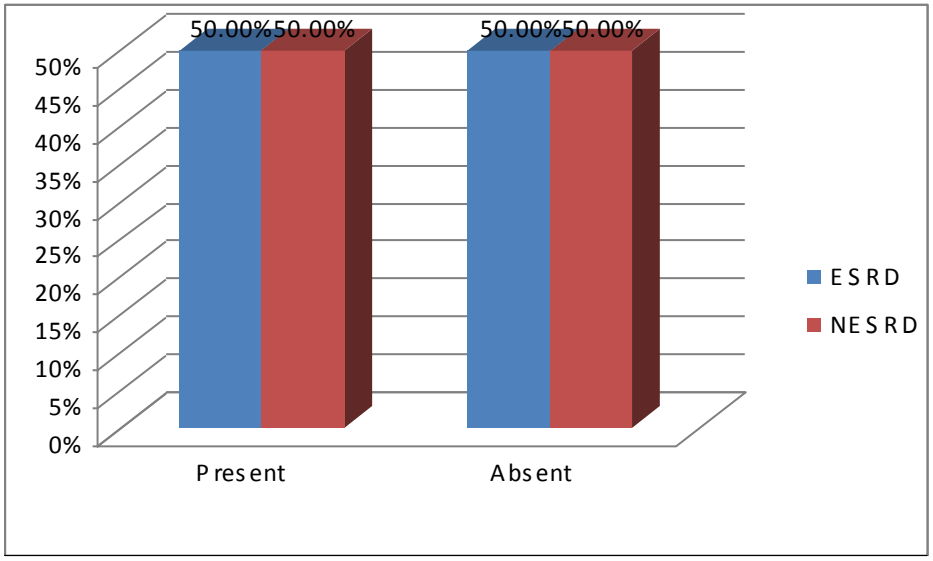
Graph 17: Distribution of ESRD and NESRD among Diabetes Mellitus patients with relation to Burning Tongue



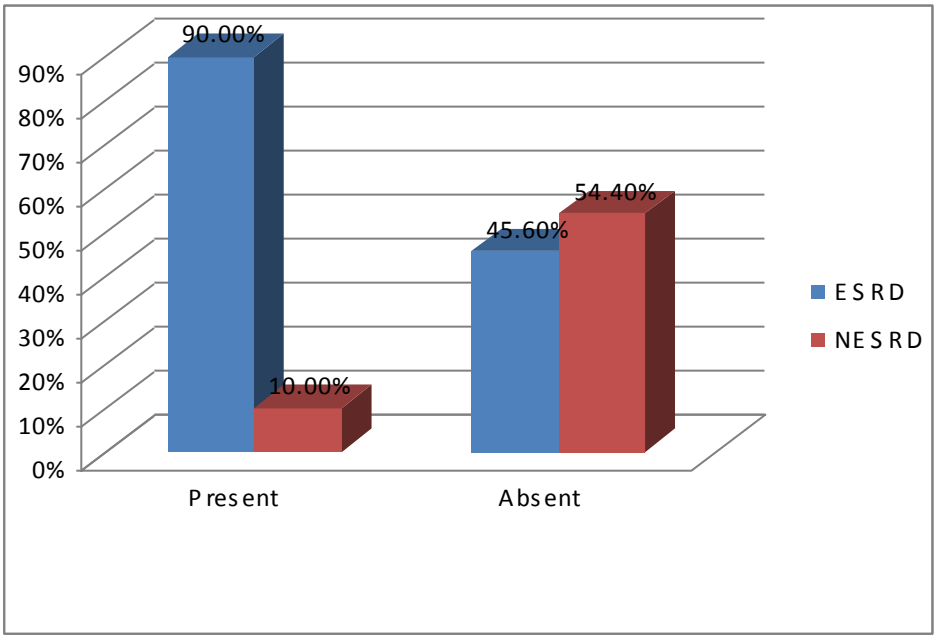
Graph 18: Distribution of ESRD and NESRD among Diabetes Mellitus patients with relation to Candidiasis



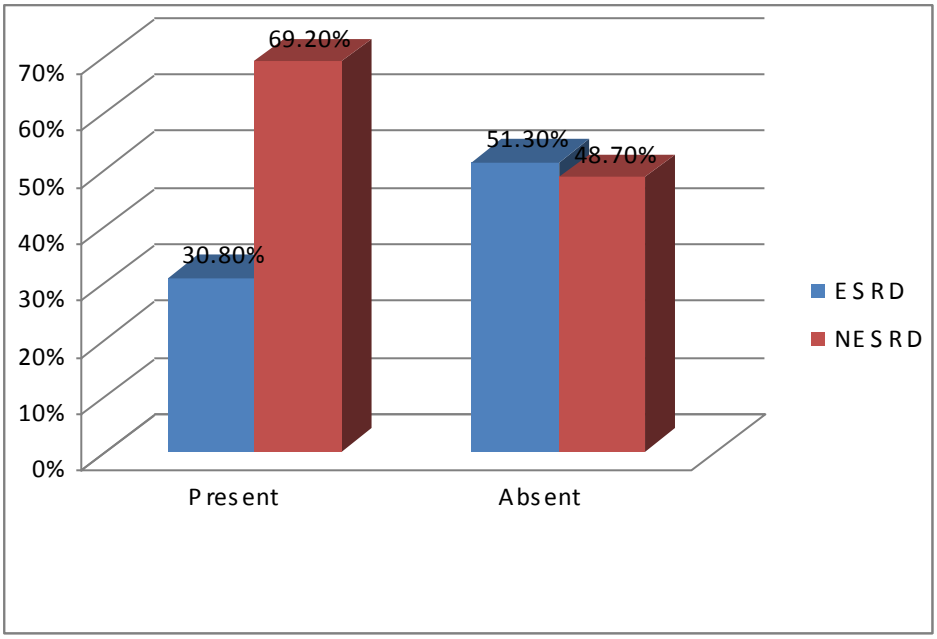
Graph 19: Distribution of ESRD and NESRD among Diabetes Mellitus patients with relation to Dry and Fissured Lips



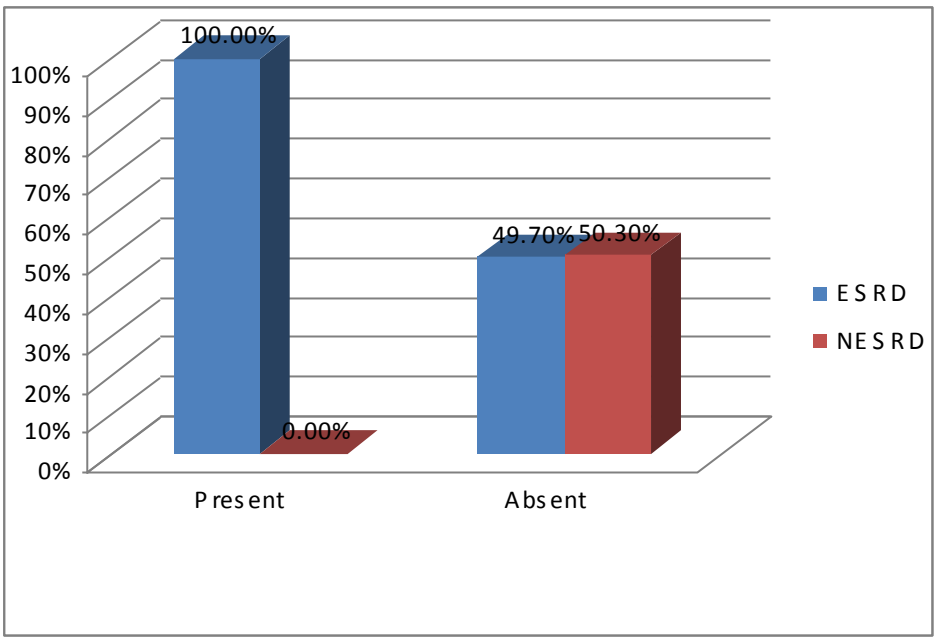
Graph 20: Distribution of ESRD and NESRD among Diabetes Mellitus patients with relation to Petechiae / Ecchymoses



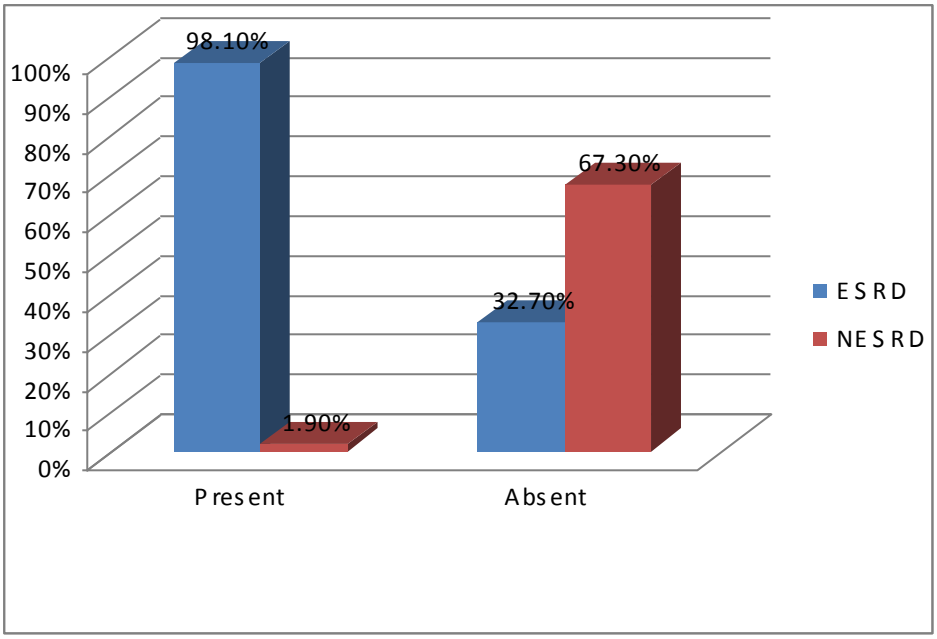
Graph 21: Distribution of ESRD and NESRD among Diabetes Mellitus patients with relation to Angular Chelitis



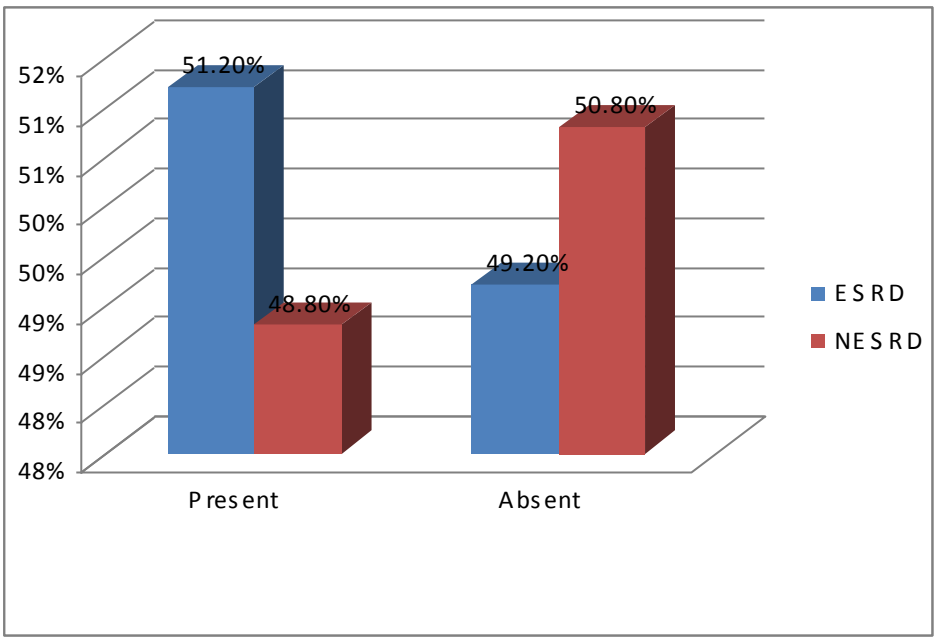
Graph 22: Distribution of ESRD and NESRD among Diabetes Mellitus patients with relation to Uremic Stomatitis



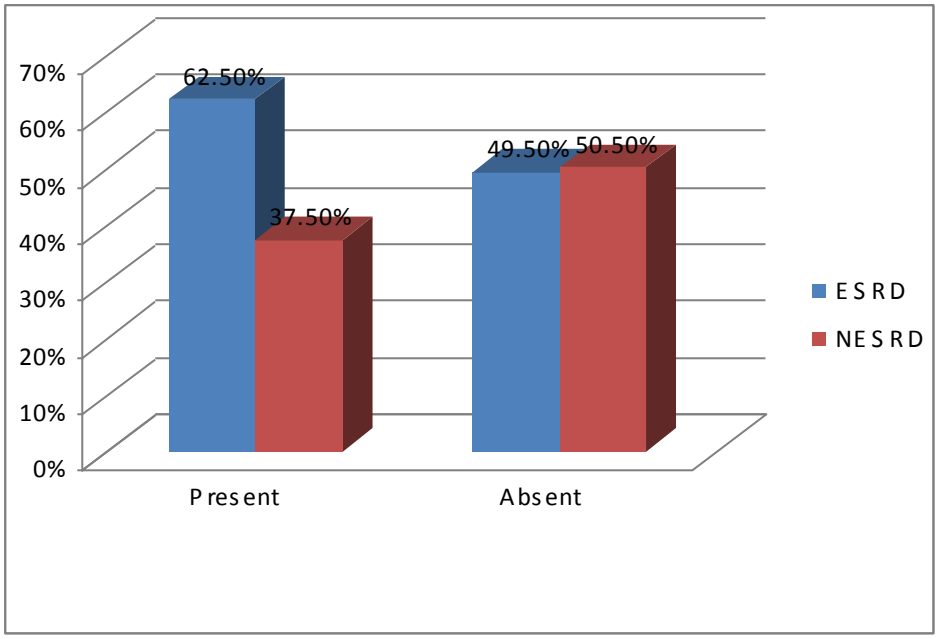
Graph 23: Distribution of ESRD and NESRD among Diabetes Mellitus patients with relation to Uremic Fetor



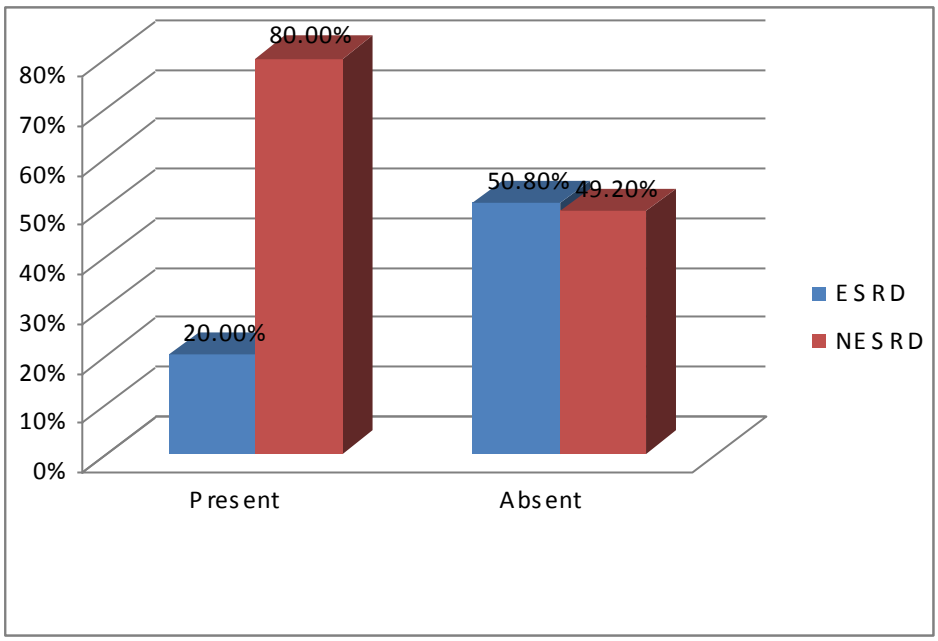
Graph 24: Distribution of ESRD and NESRD among Diabetes Mellitus patients with relation to Xerostomia



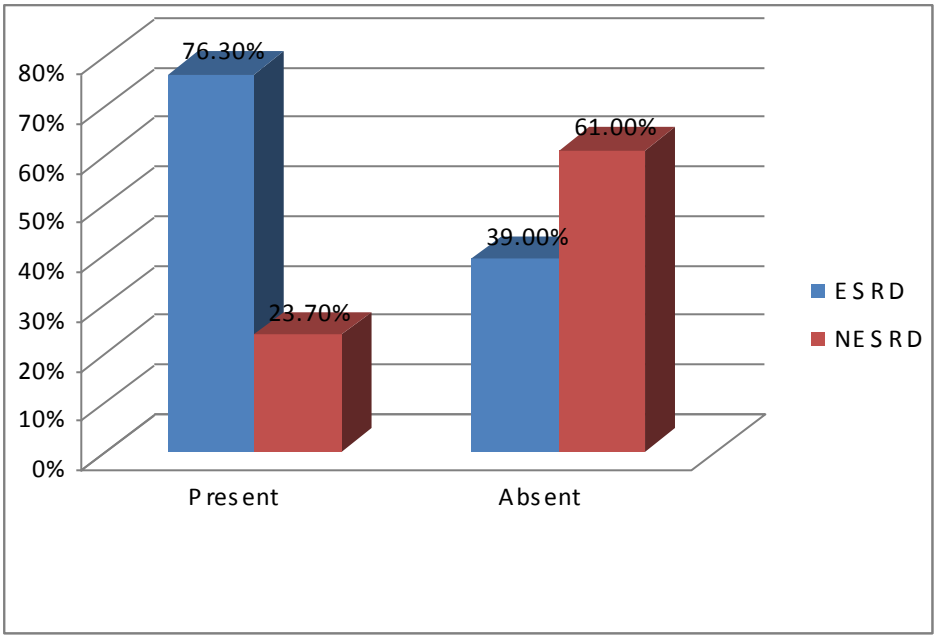
Graph 25: Distribution of ESRD and NESRD among Diabetes Mellitus patients with relation to Herpes Labialis



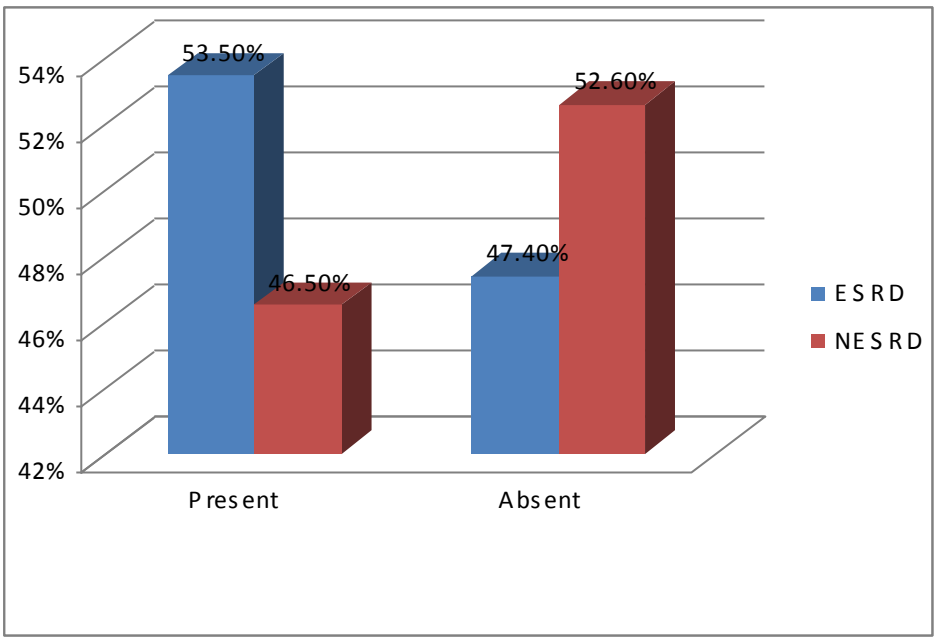
Graph 26: Distribution of ESRD and NESRD among Diabetes Mellitus patients with relation to Aphthous Ulcer



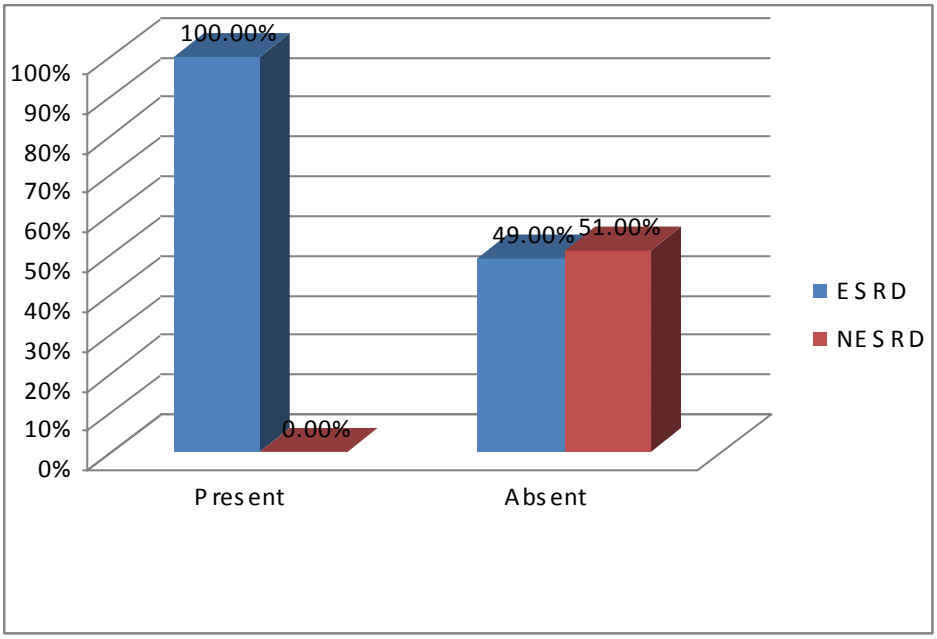
Graph 27: Distribution of ESRD and NESRD among Diabetes Mellitus patients with relation to Pale Mucosa



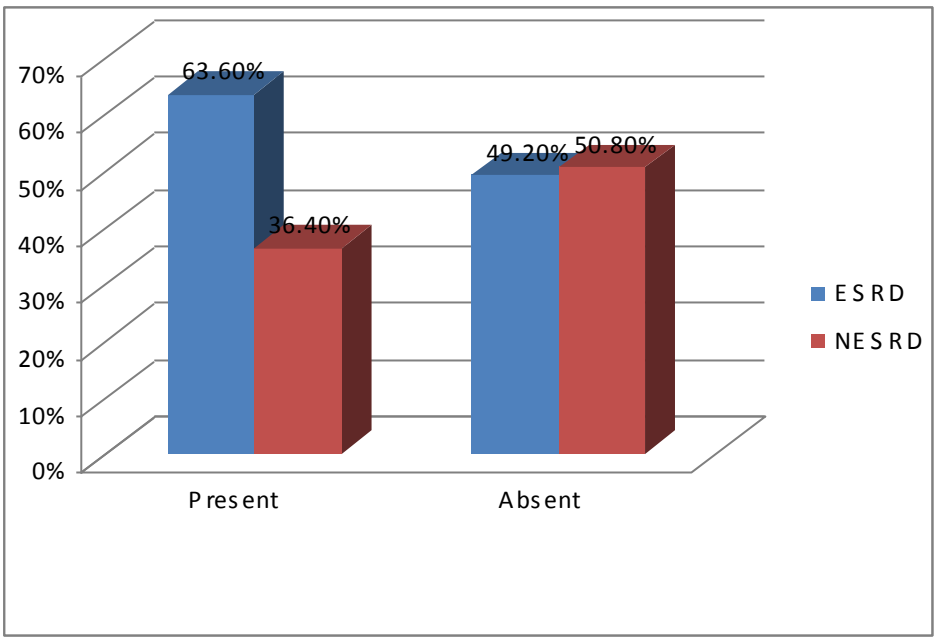
Graph 28: Distribution of ESRD and NESRD among Diabetes Mellitus patients with relation to Unpleasant Taste



Graph 29: Distribution of ESRD and NESRD among Diabetes Mellitus patients with relation to Stomatitis Medicamentosa



Graph 30: Distribution of ESRD and NESRD among Diabetes Mellitus patients with relation to Lichen Planus



Diabetic Nephropathy is becoming a major problem as the incidence of diabetes is increasing. Diabetes is currently the most common cause of renal failure in the Western World. The pathways leading to Diabetic Nephropathy are thought to involve both genetic and environmental factors intertwined in a complex fashion Glycaemic control are important for the development of Diabetic Nephropathy. This fact was established by the landmark Diabetes Control and Complications Trial (DCCT) [107,108]

The incidence of diabetes is increasing word-wide, with subsequent increase in the incidence of Diabetic Nephropathy. The earliest clinical evidence of Diabetic Nephropathy is microalbuminuria. Progression from microalbuminuria to overt nephropathy occurs in 20-40% within a 10-year period with approximately 20% of these patients progressing to end-stage renal disease. End-stage renal disease develops in 50% of type 1 diabetes patients with overt nephropathy within 10 years and in more than 75% by 20 years in the absence of treatment. In Type-2 Diabetes, a greater proportion of patients have microalbuminuria and overt nephropathy at or shortly after diagnosis of diabetes[73].

Patients with diabetes undergoing dialysis have a 22% and 15% higher mortality at one year and five years, respectively, when compared with patients without diabetes, and specifically the first-year mortality of patients with Type-2 Diabetes who require dialysis exceeds 20%[73].

Patients with macroalbuminuria, and especially those with ESRD, are at great risk of cardiovascular events and premature death. Premature

death is thus a competing outcome to ESRD in patients with diabetes and macroalbumniuria[107,108].

With respect to our study it is important not only to diferentiate between ESRD and NESRD in Diabetes Mellitus patients by oral mucosal lesion, signs and symptoms, but also to identify those with asymptomatic ESRD, who may require treatment at the earliest to prevent complications.

Investigations used in our study are

- Evaluation of Fasting Blood Sugar
- Evaluation of Hemoglobin Level
- Evaluation of Serum Creatinine
- Evaluation of Blood Urea Level.

This is in accordance with the study conducted by **De La Rosa García E et al[66]** in 2006.

This study is to assess oral signs, symptoms and oral lesions type and prevalence, in diabetic patients with End Stage Renal Disease (ESRD-DM) and compare them with analogous findings in Non-End Stage Renal Disease (NESRD-DM) group.

This study was conducted between June 2010 to May 2011 in the department of Oral Medicine and Radiology of Ragas Dental College and Hospital, and Voluntary Health Services, Adyar, Chennai

A comparative study was conducted in 200 Diabetic patients, who are diagnosed as having Non-End Stage Renal Disease and End Stage Renal

Disease undergoing dialysis will be taken up for the study. These patients will undergo general clinical examination to exclude HIV. Patients are examined for the presence of intra oral manifestations of Diabetic patients with End Stage Renal Disease (ESRD-DM) and with Non-End Stage Renal Disease (NESRD-DM) like saburral tongue, smooth tongue, burning tongue, candidiasis, dry and fissured lips, petechiae or ecchymoses, ulcerative or uremic stomatitis, herpes simplex, angular cheilitis, uremic fetor, xerostomia, pale mucosa.

STUDY ANALYSIS

ACCORDING TO SEX OF THE SUBJECTS

In the present study, among the 200 subjects, 79 patients were male and 121 patients were female. Out of the 100 ESRD patients, 41 patients (51.90%) are found to be male and 59 patients (48.80%) are found to be female .Among the 100 NESRD patients, 38 patients (48.10%) are male and 62 patients (51.20%) were female. with a P-value of $\leq$ 0.664 which is statistically not significant. Thus a negative correlation between the 2 groups with respect to sex in the distribution is seen

In our study, with respect to Sex, the female patients were more because of greater possibility of small sample size.

This is not in accordance with the study conducted by **Zohreh Hajheydari, Atieh Makhlough**[69], 43 (42.6%) women and 58 (57.4%) men , **Gall et al**[71] found that males had a 2.6 times greater risk of developing incipient or overt nephropathy, **Choy BY et al**[72] found that for ESRD

patients, the male/female ratio was reported to be about 1:1 for Diabetes Patients.

ACCORDING TO AGE GROUP

In the present study, among 200 patients, 72 were in age group of 12-40 years, 101 patients were in age group of 41-60 years, 24 patients were in age group of 61-80 years, and 3 patients were in the age group of 81-100 years. Among the 100 patients in ESRD- Diabetes Mellitus patients, 22 (30.56%) were between 12-40yrs, 59 (58.41%) were between 41- 60 yrs, 17 (70.83%) were between 61-80 yrs, 2 (66.67%) were between 81-100 yrs, Among the 100 patients in NESRD- Diabetes Mellitus patients, 50 (69.44%) were between 12- 40 yrs, 42 (41.59%) were between 41-60 yrs, 7 (29.17%) were between 61-80 yrs, 1 (33.33%) were between 81-100 yrs.

The mean age group affected by ESRD is 50.77; the mean age group affected by NESRD is 40.82, with a P-value of ≤ 0.000 which is statistically very highly significant. Thus a positive correlation between the 2 groups with respect to age in the distribution is seen..

This is in accordance with the study conducted by **Thorman R, Neovius M and Hylander B**[75], on 101 patients and found 43 (42.6%) were women and 58 (57.4%) were men with a mean age of 50.0 ± 12.3 years, **Belmiro Cavalcanti do Egito Vasconcelos et al**[28] conducted a study on 30 patients, 40% were below 60 years of age, and 60% were older than 60 years, **Chi-yuan Hsu et al**[67] found the prevalence of chronic renal insufficiency among older adults was 10-fold that of younger individuals .

However, younger individuals with chronic renal insufficiency were about 3-fold more likely to progress to ESRD.

ACCORDING TO HABIT AMONG THE STUDY SUBJECTS

In the present study, among the 200 patients, 35 patients have smoking habits, and 24 patients have tobacco related habits. Among 100 ESRD patients, 22 (62.90%) patients have smoking habits, and 18 (75%) patients have tobacco related habits. Among 100 NESRD patients, 13 (37.10%) patients have smoking habits, and 6 (25%) patients have tobacco related habits, with a P-value of ≤ 0.003 which is statistically highly significant. Thus a positive correlation between the 2 groups with respect to habits in the distribution is seen

This is in accordance with the study conducted by **Olugbenga. E. Ayodele et al**[73], found that Smoking with a P-value ≤ 0.001, causes a substantial increase in the risk of both micro- and macrovascular diseases in Diabetes. Smoking is an independent risk factor for the development of Diabetic Nephropathy and is associated with an accelerated loss of renal function, an increased risk for ESRD, and decreased survival on commencement of dialysis. Loss of renal function is slower in those who stopped smoking. Cessation of smoking alone may reduce the risk of progression by 30% in patients with Type-2 Diabetes

ACCORDING TO DIET AMONG THE STUDY SUBJECTS

In the present study, among the 200 patients, 106 patients were on renal diet, 91 patients were on vegetarian diet and 3 patients were on Non

vegetarian diet. Among 100 ESRD patients, 29 patients (27.4%) were on renal diet, 68 patients (74.70%) were on vegetarian diet and 3 (100%) patients were on Non vegetarian diet. Among 100 NESRD patients, 77 patients (72.6%) were on renal diet, 23 patients (25.30%) were on vegetarian diet and 0 (0%) patients were on Non vegetarian diet.

With a P-value of ≤ 0.000 which is statistically very highly significant, a positive correlation between the 2 groups with respect to habits in the distribution is seen

This is in accordance with the study conducted by **Jorge l Gross et al** [74] in 108 patients, that dietary protein restriction slowed the progression of Diabetic Nephropathy in patients with type 1 diabetes. More recently, a 4-year randomized controlled trial in 82 patients with type 1 diabetes with progressive Diabetic Nephropathy showed that a moderately low–protein diet (0.9 g / kg / day) reduced the risk of end-stage renal disease or death by 76%.

ACCORDING TO TYPE OF DIABETES MELLITUS **AMONG THE STUDY SUBJECTS**

In this present study, among the 200 patients, 39 patients were IDDM (Insulin– Dependent Diabetes Mellitus) and 161 patients were NIDDM (Non Insulin – Dependent Diabetes Mellitus). Among 100 ESRD patients, 17 (43.6%) were IDDM (Insulin – Dependent Diabetes Mellitus) patients and 83 (51.60%) were NIDDM (Non Insulin – Dependent Diabetes Mellitus) patients. Among 100 NESRD patients, 22 (56.4%) were IDDM

(Insulin – Dependent Diabetes Mellitus) patients and 78 (48.4%) were NIDDM (Non Insulin – Dependent Diabetes Mellitus) patients, with a P-value of ≥ 0.372 which is statistically not significant. Thus a negative correlation between the 2 groups with respect to type of Diabetes Mellitus in the distribution is seen

In our study, with respect to type of Diabetes Mellitus, the NIDDM (Non Insulin – Dependant Diabetes Mellitus) patients were affected more with ESRD.

This is in accordance with the study conducted by **Ritz E, Bergis K, Strojek K and Keller C**[78], found that Diabetic Nephropathy in patients with Type II Diabetes has become the most frequent cause of End stage Renal Failure in Germany.

ACCORDING TO DURATION OF DIABETES MELLITUS **AMONG THE STUDY SUBJECTS**

In this present study, among the 200 patients ,140 were found to be less than 12 yrs, 59 were between 12-40yrs, 1 (100%) were between 40- 60 yrs, 0 (0%) were between 60-80 yrs. Among the 100 patients in ESRD-Diabetes Mellitus patients, 54 (38.58%) were found to be less than 12 yrs, 45 (76.28%) were between 12-40yrs, 1 (100%) were between 40- 60 yrs, 0 (0%) were between 60-80 yrs. Among the 100 patients in NESRD- Diabetes Mellitus patients, 86 (61.42%) were found to be less than 12 yrs, 14 (23.72%) were between 12- 40 yrs, 0 (0%) were between 40-60 yrs, 0 (0%) were between 60-80 yrs, with a P-value of ≥ 0.000 which is statistically very

highly significant. Thus a positive correlation between the 2 groups with respect to duration of Diabetes Mellitus in the distribution is seen

In our study, with respect to duration of Diabetes Mellitus, the mean duration of Diabetes Mellitus age group affected by ESRD is 14.39 years and the mean duration of Diabetes Mellitus group affected by NESRD is 5.82 years.

This is in accordance with the study conducted by **Gábor L. Kovács**[76] in which he found that patients who have Type 1 diabetes with nephropathy and hypertension, 50% will go on to develop end-stage renal disease within 10 years. 80% of people who have Type 1 diabetes and microalbuminuria will progress to overt nephropathy, whereas only 20-40% of those with Type 2 Diabetes over a period of 15 years will progress.

E. de la Rosa García et al [93] conducted a study on 233 patients, In 133 ESRD DM patients, the median known duration of diabetes before dialysis was 17 years.

ACCORDING TO FAMILY HISTORY DIABETES MELLITUS AMONG THE STUDY SUBJECTS

In this present study, among the 200 patients, 46 patients had family history of Diabetes Mellitus. Among 100 ESRD patients, 24 (52.2%) had family history of Diabetes Mellitus. Among 100 NESRD patients, 22 (47.8%) had family history of Diabetes Mellitus, with a P-value of ≥ 0.737 which is statistically not significant. Thus a negative correlation between the

2 groups with respect to family history of Diabetes Mellitus in the distribution is seen

This is not in accordance with the study conducted by **Barry I Freedman et al** [79] Family histories were obtained from 4365 dialysis patients (83% of those eligible), and 856 (20%) reported having a family history of ESRD.

ACCORDING TO HYPERTENSION AMONG THE STUDY SUBJECTS:

In this present study, among the 200 patients, 40 patients were hypertensive. Among 100 ESRD patients, 26 (65%) were hypertensive patients. Among 100 NESRD patients, 14 (35%) were hypertensive patients. with a P-value of ≥ 0.030 which is statistically significant. Thus a positive correlation between the 2 groups with respect to hypertension in the distribution is seen

This is in accordance with the study conducted by **Britt B. Newsome et al** [77], on 87,094 patients, found the history of hypertension (P-value is 0.01) was statistically significant.

ACCORDING TO FASTING BLOOD SUGAR LEVEL AMONG THE STUDY SUBJECTS:

In this present study, among the 200 patients,71 were found to be less than 120 mg/dl, 79 were between 120-200 mg/dl, 50 were more than 200 mg/dl. Among the 100 patients in ESRD- Diabetes Mellitus patients, 30 (42.25%) were found to be less than 120 mg/dl, 42 (53.17%) were between

120-200 mg/dl, 28 (56%) were more than 200 mg/dl. Among the 100 patients in NESRD- Diabetes Mellitus patients, 41 (57.75%) were found to be less than 120 mg/dl, 37 (46.83%) were between 120-200 mg/dl, 22 (44%) were more than 200 mg/dl, with a P-value of ≥ 0.330 which is statistically not significant. Thus a negative correlation between the 2 groups with respect to fasting blood sugar level in the distribution is seen

The mean value for the fasting blood sugar level in ESRD patients is 161.63mg/dl. The mean value for the fasting blood sugar level in NESRD patients is 153.18mg/dl

This is not in accordance with the study conducted by **Vesterinen M et al**[89], in CKD patients with Diabetes Mellitus had poor glycemic control as expected (mean HbA1C) 8.0% the normal value being ≤ 5.9%, with a P-value <0.01.

ACCORDING TO HEMOGLOBIN LEVEL AMONG THE STUDY SUBJECTS

In this present study, among the 200 patients 63 were found to be less than 10 mg/dl, 128 were between 10-15 mg/dl, 9 were more than 15 mg/dl.Among the 100 patients in ESRD- Diabetes Mellitus patients, 56 (88.89%) were found to be less than 10 mg/dl, 41 (32.03%) were between 10-15 mg/dl, 3 (33.33%) were more than 15 mg/dl. Among the 100 patients in NESRD- Diabetes Mellitus patients, 7 (11.11%) were found to be less than 10 mg/dl, 87 (67.97%) were between 10-15 mg/dl, 6 (66.67%) were more than 15 mg/dl. with a P-value of ≥ 0.000 which is statistically very

highly significant. Thus a positive correlation between the 2 groups with respect to hemoglobin level in the distribution is seen

The mean value for the hemoglobin level in ESRD patients is 9.81mg/dl. The mean value for the hemoglobin level in NESRD patients is 12.3mg/dl

This is in accordance with the study conducted by **Francois Madore et Al**[70] found variables of nutritional status in terms of serum albumin and Creatinine concentration, and the dose of dialysis -urea reduction ratio were found to be significantly associated with Hemoglobin concentration ($P < 0.001$). Age, race, and gender were also found to be significantly associated with haemoglobin concentrations ($P < 0.001$) and concluded that Anemia may be predictive of an increased risk of mortality in some hemodialysis patients.

ACCORDING TO SERUM CREATININE LEVEL AMONG THE STUDY SUBJECTS

In this present study, among the 200 patients, 100 were found to be less than 2 mg/dl, 91 were between 2-10 mg/dl, 9 were more than 10 mg/dl. Among the 100 patients in ESRD- Diabetes Mellitus patients, 0 (0%) were found to be less than 2 mg/dl, 91 (100%) were between 2-10 mg/dl, 9 (100%) were more than 10 mg/dl. Among the 100 patients in NESRD-Diabetes Mellitus patients, 100 (100%) were found to be less than 2 mg/dl, (0%) were between 2-10 mg/dl, 0 (0%) were more than 10 mg/dl. with a P-value of ≥ 0.000 which is statistically very highly significant. Thus a

positive correlation between the 2 groups with respect to concentration of serum creatinine level in the distribution is seen

The mean value for the Serum Creatinine in ESRD patients is 4.334mg/dl. The mean value for the Serum Creatinine in NESRD patients is 0.945mg/dl.

This is in accordance with the study conducted by **De La Rosa García E et al[66]** in 2006, evaluated 229 individuals, Group A: ESRD DM on dialysis, and group B: Non-ESRD DM categorized as patients with serum creatinine <2.0 mg/dl.

Sanjay Kumar Agarwal et al[51] who, stated that serum creatinine persistently >1.8mg% for 8–12 weeks in the absence of any reversible factor was the criterion to diagnose Chronic Renal Failure, **Agarwal et al**[84] screened 4900 persons in urban communities of Delhi and found a .79% point prevalence of persons with Serum Creatinine more than1.8 mg/dL.

A M El Nahas et al[83] stated that the ratio of plasma urea to creatinine concentration accurately reflected the dietary protein intake: it rose to 110 during the high protein diet and subsequently fell to 40 during the low protein diet with a P-value of <0 025.

ACCORDING TO BLOOD UREA LEVEL AMONG THE STUDY SUBJECTS

In this present study, among the 200 patients, 137 were found to be less than 50 mg/dl, 40 were between 50-100 mg/dl, 23 (100%) were more than 100 mg/dl. Among the 100 patients in ESRD- Diabetes Mellitus

patients, 37 (27%) were found to be less than 50 mg/dl, 40 (100%) were between 50-100 mg/dl, 23 (100%) were more than 100 mg/dl. Among the 100 patients in NESRD- Diabetes Mellitus patients, 100 (73%) were found to be less than 50 mg/dl, 0 (0%) were between 50-100 mg/dl, 0 (0%) were more than 100 mg/dl. with a P-value of ≥ 0.000 which is statistically very highly significant. Thus a positive correlation between the 2 groups with respect to concentration of blood urea level in the distribution is seen

The mean value for the blood urea level in ESRD patients is 73.72mg/dl. The mean value for the blood urea level in NESRD patients is 28.66mg/dl

There were no studies with regard to blood urea level in ESRD and NESRD - Diabetes Mellitus patients to correlate with the findings of our study.

ACCORDING TO MEDICATION FOR DIABETES MELLITUS **AMONG THE STUDY SUBJECTS**

In this study group, among 200 subjects, 58 patients had taken metformin, 95 patients had taken sulphonyl urea and 47 patients had taken insulin. Out of the 100 ESRD patients, 52 patients (89.70%) had taken metformin, 40 (42.10%) patients had taken sulphonyl urea and 8 (17%) patients had taken insulin. Among 100 NESRD patients, 6 patients (10.30%) had taken metformin, 55 (57.90%) patients had taken sulphonyl urea and 39 (83%) patients had taken insulin, with a P-value of ≥ 0.000 which is statistically very highly significant. Thus a positive correlation between the

2 groups with respect to medication for Diabetes Mellitus in the distribution is seen.

In this present study also patients who have been having serum creatinine level ≥ 2.0 mg /dl have been under the medication of metformin, and sulphonyl urea

This is in accordance with the study conducted by **Jorge l. Gross et al[74]**. They stated that Metformin should not be used when Serum Creatinine is >1.5 mg/dl in men and >1.4 mg/dl in women due to the increased risk of lactic acidosis. Sulfonylureas and their metabolites except glimepiride, are eliminated via renal excretion and should not be used in patients with decreased renal function Thus, most type 2 diabetic patients with Diabetic Nephropathy should be treated with insulin.

Janelle C Nisbet et al[97], found that life-threatening lactic acidosis can occur, caused by accumulation of metformin, and that risk factors for this include renal impairment, old age and doses over 2 g per day. . The estimated prevalence of life-threatening lactic acidosis is one to five cases per 1, 00,000 with mortality in reported cases up to 50%.

Devasmita Choudhury et al[98]., suggested the Use of metformin and the first-generation sulfonylurea agents chlorpropamide, tolbutamide and tolazamide, as well as the α-glucosidase inhibitors acarbose and miglitol, should be avoided in patients with advanced CKD or ESRD, in light of their association with metabolic acidosis and prolonged hypoglycemia.

Abe M, et al[94] stated that Conventional oral hypoglycemic agents, such as sulfonylurea (SU), are not suitable due to the risk of prolonged hypoglycemia; furthermore, metformin is contraindicated for moderate to advanced CKD,

Jean-Franc¸ois Yale[95], found that Insulin also can be used safely in renal failure

THE PREVALANCE OF ORAL MUCOSAL LESIONS AMONG THE STUDY SUBJECTS

In this present study, among the 200 patients, 24 patients had Saburral Tongue (p value ≤ 0.002), 4 patients had Fissured Tongue (P value ≤ 1.000), 18 patients had smooth Tongue (P value ≥ 0.323), 23 patients had burning Tongue (P value ≥ 0.506), 51 patients had Candidiasis (P value ≥ 0.417), 44 patients had Dry and Fissured Lips (P value ≥ 1.000), 20 patients had Petechiae / Ecchymoses (P value ≤ 0.000), 13 patients had Angular Chelitis (P value ≥ 0.152) , 1 patients had Uremic Stomatitis (P value ≥ 0.316), 53 patients had Uremic Fetor (P value ≤ 0.000), 82 patients had Xerostomia (P value ≥ 0.774), 8 patients had Herpes Labialis (P value ≥ 0.470), 5 patients had Aphthous Ulcer (P value ≥ 0.174), 59 patients had Pale Mucosa (P value ≤ 0.000), 86 patients had Unpleasant Taste (P value ≥ 0.391), 4 patients had Stomatitis Medicamentosa (P value ≤ 0.043), 11 patients had Lichen Planus (P value ≥ 0.352).

Among 100 ESRD patients, 19 (79.20%) had Saburral Tongue, 2 (50%) had Fissured Tongue, 11 (61.10%) had smooth Tongue, 13 (56.50%)

had Burning Tongue, , 23 (45.10%) had Candidiasis, 22 (50.00%) had Dry and Fissured Lips, 18 (90.00%) had Petechiae / Ecchymoses, 4 (30.80%) had Angular Chelitis, , 1(100%) patients had Uremic Stomatitis, 52 (98.10%) had Uremic Fetor, 42 (51.20%) had Xerostomia 5 (62.50%) had Herpes Labialis, 1 (20%) had Aphthous Ulcer, 45 (76.30%) had Pale Mucosa, 46 (53.50%) had Unpleasant Taste, 4 (100%) had Stomatitis Medicamentosa, 7 (63.60%) had Lichen Planus.

Among 100 NESRD patients, 5 (20.80%) had Saburral Tongue, 2 (50%) had Fissured Tongue, 7 (38.90%) had smooth Tongue, 10 (43.50%) had Burning Tongue, 28 (54.90%) had Candidiasis, 22 (50.00%) had Dry and Fissured Lips, 2 (10.00%) had Petechiae / Ecchymoses, 9 (69.20%) had Angular Chelitis, 0 (0%) patients had Uremic Stomatitis , 1 (1.90%) had Uremic Fetor, 40 (48.80%) had Xerostomia, 3 (37.50%) had Herpes Labialis, 4 (80%) had Aphthous Ulcer, 14 (23.70%) had Pale Mucosa, 40 (46.50%) had Unpleasant Taste , 0 (0%) had Stomatitis Medicamentosa, 4 (36.40%) had Lichen Planus.

This is in accordance with the study conducted by **Shu-Fen Chuang et al**[81] in which they found that the incidence of uremic odor in CRF patients with Diabetes Mellitus (27.9%). Incidence of mucosal petechia / ecchymosis was 20.9% in CRF patients with Diabetes Mellitus.

De La Rosa García E et al[66] evaluated 229 individuals,. Two adult groups were studied: Group A: ESRD DM on dialysis, and group B: non-ESRD DM (Serum Creatinine <2.0 mg/dl). group A 99, and group B 130

pts. Signs and symptoms prevalence was higher in group A: uremic breath (48.5%), unpleasant taste (45.5%) and Xerostomia (44.4%) being the most frequent ones. Oral Lesions were also more prevalent in group A. The most frequent Oral Lesions were dry, fissured lips (28.3%), saburral tongue (18.2%) and candidiasis (17.2%). No difference was found in candidiasis prevalence between groups. Candidiasis was found associated to xerostomia and smooth tongue only in group A. The high prevalence of uremic fetor, xerostomia, saburral tongue and candidiasis in group A, could be tried as warning signs on the possibility of non diagnosed advanced renal disease in other diabetic patients,

De la Rosa-García E et al[91], performed a study in 90 patients Saburral tongue (ST) was found in 22% of the patients.

P. Mosannen Mozaffari et al[82] stated that, one of the early symptoms may be a bad metallic taste and unpleasant odor in the mouth particularly in the morning. This uremic fetor, an ammoniacal odor is a typical sign of all uremic patient. Four of 300 patients with uremia were observed to have probable uremic stomatitis in the 1930s, while in 1964 another 4 affected patients were reported from a group of 262 patients with renal disease

But not in accordance with study conducted by **Neovius M and Hylander B et al**[75], on 101 patients and found oral fungal infection (OFI) was found in 32% of the ESRD patients and 11% of the controls (P=0.007), angular cheilitis, were found in 37% of the patients with OFI (P=0.0002).

Udayakumar P et al [80] conducted a study on One hundred patients with CRF on hemodialysis and found Oral changes included macroglossia with teeth markings (35%), xerostomia (31%), ulcerative stomatitis (29%), angular cheilitis (12%) and uremic breath (8%). Ulcerative stomatitis seen in 29% is reported to occur in patients with blood urea level more than 150mg/ml.

Sowell SB[86], Carl W and Wood RH [87], Hovinga J et al[88] found Uremic stomatitis is often a clinical finding in cases of advanced disease.

Gavaldá et al[90] examined the oral mucosa of individuals with chronic renal failure and noted several mucosal lesions, uremic stomatitis and Candida infections in 37% of these patients

Thorman R et al[75] evaluated 93 ESRD patients and 45 age- and gender-matched controls OFI was found in 32% of the ESRD patients and 11% of the controls (P-value is 0.007),

Safia A. Al-Attas et al[92] conducted a study on 150 Diabetics. They found that the number of patients with candidal carriage from the oral cavity was higher in patients with type 1 diabetes than in type 2 (*P*=.003),

E. de la Rosa García et al[93] conducted a study on ESRD DM and DM groups, in that order, consisting of 103 and 130 patients respectively 45.6% and 26.9% (p = 0.003) reported unpleasant taste, and dry mouth (P-value is 0.011).

This study is to assess oral signs, symptoms and oral lesions type and prevalence, in diabetic patients with End Stage Renal Disease (ESRD-DM) and compare them with analogous findings in Non-End Stage Renal Disease (NESRD-DM) group.

This study was conducted between June 2010 to May 2011 in the department of Oral Medicine and Radiology of Ragas Dental College and Hospital, and Voluntary Health Services, Adyar, Chennai

The 200 Diabetic patients, who are diagnosed as having Non-End Stage Renal Disease (serum creatinine less than 2mg/dl) and End Stage Renal Disease undergoing dialysis will be taken up for the study. These patients will undergo general clinical examination to exclude HIV. The subjects were made to sit comfortably on a Dental Chair. Sterile hand gloves were used during examination of the patients. Patients were examined under halogen lamp in the dental chair under aseptic conditions and relevant demographic data were collected. Patients are examined for the presence of intra oral manifestations

The study documents the following data:

- In the present study, among the 200 subjects, 79 patients were male and 121 patients were female. Out of the 100 ESRD patients, 41 patients (51.90%) are found to be male and 59 patients (48.80%) are found to be female .Among the 100 NESRD patients, 38 patients (48.10%) are male and 62 patients (51.20%) were female

- Among 200 patients, 72 were in age group of 12-40 years, 101 patients were in age group of 41-60 years, 24 patients were in age group of 61-80 years, and 3 patients were in the age group of 81-100 years. Among the 100 patients in ESRD- Diabetes Mellitus patients, 22 (30.56%) were between 12-40yrs, 59 (58.41%) were between 41-60 yrs, 17 (70.83%) were between 61-80 yrs, 2 (66.67%) were between 81-100 yrs, Among the 100 patients in NESRD- Diabetes Mellitus patients, 50 (69.44%) were between 12- 40 yrs, 42 (41.59%) were between 41-60 yrs, 7 (29.17%) were between 61-80 yrs, 1 (33.33%) were between 81-100 yrs.
- Among the 200 patients, 141 patients did not have any habits, 35 patients have smoking habits, and 24 patients have tobacco related habits. Among 100 ESRD patients, 60 (42.60%) patients did not have any habits, 22 (62.90%) patients have smoking habits, and 18 (75%) patients have tobacco related habits. Among 100 NESRD patients, 81 (57.40%) patients did not have any habits, 13 (37.10%) patients have smoking habits, and 6 (25%) patients have tobacco related habits.
- Among the 200 patients, 106 patients were on renal diet, 91 patients were on vegetarian diet and 3 patients were on Non vegetarian diet. Among 100 ESRD patients, 29 patients (27.4%) were on renal diet, 68 patients (74.70%) were on vegetarian diet and 3 (100%) patients were on Non vegetarian diet. Among 100 NESRD patients, 77

patients (72.6%) were on renal diet, 23 patients (25.30%) were on vegetarian diet and 0 (0%) patients were on Non vegetarian diet.

- Among the 200 patients, 39 patients were IDDM (Insulin–Dependent Diabetes Mellitus) and 161 patients were NIDDM (Non Insulin – Dependent Diabetes Mellitus). Among 100 ESRD patients, 17 (43.6%) were IDDM (Insulin – Dependent Diabetes Mellitus) patients and 83 (51.60%) were NIDDM (Non Insulin – Dependent Diabetes Mellitus) patients. Among 100 NESRD patients, 22 (56.4%) were IDDM (Insulin – Dependent Diabetes Mellitus) patients and 78 (48.4%) were NIDDM (Non Insulin – Dependent Diabetes Mellitus) patients.
- Among the 200 patients, 140 were found to be less than 12 yrs, 59 were between 12-40yrs, 1 (100%) were between 40- 60 yrs, 0 (0%) were between 60-80 yrs. Among the 100 patients in ESRD- Diabetes Mellitus patients, 54 (38.58%) were found to be less than 12 yrs, 45 (76.28%) were between 12-40yrs, 1 (100%) were between 40- 60 yrs, 0 (0%) were between 60-80 yrs. Among the 100 patients in NESRD- Diabetes Mellitus patients, 86 (61.42%) were found to be less than 12 yrs, 14 (23.72%) were between 12- 40 yrs, 0 (0%) were between 40-60 yrs, 0 (0%) were between 60-80 yrs
- Among the 200 patients, 46 patients had family history of Diabetes Mellitus and 154 patients did not have any family history of Diabetic Mellitus. Among 100 ESRD patients, 24 (52.2%) had family history

of Diabetes Mellitus and 76 (49.40%) patients did not have any family history of Diabetes Mellitus. Among 100 NESRD patients, 22 (47.8%) had family history of Diabetes Mellitus and 78 (50.60%) patients did not have any family history of Diabetes Mellitus.

- The fasting blood sugar level was found among the 200 patients, 71 were found to be less than 120 mg/dl, 79 were between 120-200 mg/dl, and 50 were more than 200 mg/dl. Among the 100 patients in ESRD- Diabetes Mellitus patients, 30 (42.25%) were found to be less than 120 mg/dl, 42 (53.17%) were between 120-200 mg/dl, 28 (56%) were more than 200 mg/dl. Among the 100 patients in NESRD- Diabetes Mellitus patients, 41 (57.75%) were found to be less than 120 mg/dl, 37 (46.83%) were between 120-200 mg/dl, 22 (44%) were more than 200 mg/dl,
- Among the 200 patients, 40 patients were hypertensive and 160 patients were normotensive. Among 100 ESRD patients, 26 (65%) were hypertensive patients and 74 (46.30%) were normotensive. Among 100 NESRD patients, 14 (35%) were hypertensive patients and 86 (53.70%) were normotensive
- The hemoglobin level was found among the 200 patients, 63 were found to be less than 10 mg/dl, 128 were between 10-15 mg/dl, 9 were more than 15 mg/dl.Among the 100 patients in ESRD-Diabetes Mellitus patients, 56 (88.89%) were found to be less than 10 mg/dl, 41 (32.03%) were between 10-15 mg/dl, 3 (33.33%) were

more than 15 mg/dl. Among the 100 patients in NESRD- Diabetes Mellitus patients, 7 (11.11%) were found to be less than 10 mg/dl, 87 (67.97%) were between 10-15 mg/dl, 6 (66.67%) were more than 15 mg/dl.

- The Serum Creatinine level was found among the 200 patients, 100 were found to be less than 2 mg/dl, 91 were between 2-10 mg/dl, 9 were more than 10 mg/dl. Among the 100 patients in ESRD- Diabetes Mellitus patients, 0 (0%) were found to be less than 2 mg/dl, 91 (100%) were between 2-10 mg/dl, 9 (100%) were more than 10 mg/dl. Among the 100 patients in NESRD - Diabetes Mellitus patients, 100 (100%) were found to be less than 2 mg/dl, (0%) were between 2-10 mg/dl, 0 (0%) were more than 10 mg/dl.
- The blood urea level was found among the 200 patients, 137 were found to be less than 50 mg/dl, 40 were between 50-100 mg/dl, 23 (100%) were more than 100 mg/dl. Among the 100 patients in ESRD- Diabetes Mellitus patients, 37 (27%) were found to be less than 50 mg/dl, 40 (100%) were between 50-100 mg/dl, 23 (100%) were more than 100 mg/dl. Among the 100 patients in NESRD- Diabetes Mellitus patients, 100 (73%) were found to be less than 50 mg/dl, 0 (0%) were between 50-100 mg/dl, 0 (0%) were more than 100 mg/dl.
- Among 200 subjects, 58 patients had taken metformin, 95 patients had taken sulphonyl urea and 47 patients had taken insulin. Out of

the 100 ESRD patients, 52 patients (89.70%) had taken metformin, 40 (42.10%) patients had taken sulphonyl urea and 8 (17%) patients had taken insulin. Among 100 NESRD patients, 6 patients (10.30%) had taken metformin, 55 (57.90%) patients had taken sulphonyl urea and 39 (83%) patients had taken insulin,

- Among the 200 patients, 24 patients had Saburral Tongue (p value ≤ 0.002), 4 patients had Fissured Tongue(p value ≤ 1.000), 18 patients had smooth Tongue (P value ≥ 0.323), 23 patients had burning Tongue (P value ≥ 0.506), 51 patients had Candidiasis (P value ≥ 0.417), 44 patients had Dry and Fissured Lips (P value ≥ 1.000), 20 patients had Petechiae / Ecchymoses (P value ≤ 0.000), 13 patients had Angular Chelitis (P value ≥ 0.152) , 1 patients had Uremic Stomatitis (P value ≥ 0.316), 53 patients had Uremic Fetor (P value ≤ 0.000), 82 patients had Xerostomia (P value ≥ 0.774), 8 patients had Herpes Labialis (P value ≥ 0.470), 5 patients had Aphthous Ulcer (P value ≥ 0.174), 59 patients had Pale Mucosa (P value ≤ 0.000), 86 patients had Unpleasant Taste (P value ≥ 0.391), 4 patients had Stomatitis Medicamentosa (P value ≤ 0.043), 11 patients had Lichen Planus (P value ≥ 0.352). Among 100 ESRD patients, 19 (79.20%) had Saburral Tongue, 2 (50%) had Fissured Tongue, 11 (61.10%) had smooth Tongue, 13 (56.50%) had Burning Tongue, , 23 (45.10%) had Candidiasis, 22 (50.00%) had Dry and Fissured Lips, 18 (90.00%) had Petechiae / Ecchymoses, 4 (30.80%)

had Angular Chelitis, , 1(100%) patients had Uremic Stomatitis, 52 (98.10%) had Uremic Fetor, 42 (51.20%) had Xerostomia 5 (62.50%) had Herpes Labialis, 1 (20%) had Aphthous Ulcer, 45 (76.30%) had Pale Mucosa, 46 (53.50%) had Unpleasant Taste, 4 (100%) had Stomatitis Medicamentosa, 7 (63.60%) had Lichen Planus. Among 100 NESRD patients, 5 (20.80%) had Saburral Tongue, 2 (50%) had Fissured Tongue, 7 (38.90%) had smooth Tongue, 10 (43.50%) had Burning Tongue, 28 (54.90%) had Candidiasis, 22 (50.00%) had Dry and Fissured Lips, 2 (10.00%) had Petechiae / Ecchymoses, 9 (69.20%) had Angular Chelitis, 0 (0%) patients had Uremic Stomatitis , 1 (1.90%) had Uremic Fetor, 40 (48.80%) had Xerostomia, 3 (37.50%) had Herpes Labialis, 4 (80%) had Aphthous Ulcer, 14 (23.70%) had Pale Mucosa, 40 (46.50%) had Unpleasant Taste ,), 0 (0%) had Stomatitis Medicamentosa 4 (36.40%) had Lichen Planus.

In conclusion of the study it is to be pointed out that ESRD-DM patients had a significantly higher prevalence of signs, symptoms and oral lesions, as compared to non-ESRD DM patients. This observation agrees with previous reports of ESRD predisposing to oral manifestations in the diabetic patient. Oral manifestations were, moreover, barely symptomatic when present, or were probably less of a trouble for the patient as compared to other manifestations of ESRD. Those frequently found conditions of uremic fetor, unpleasant taste, xerostomia, pale mucosa, burning tongue,

dry- fissured lips, candidiasis, saburral tongue or smooth tongue in our study group, could be tried as warning signs for undiagnosed kidney disease in other diabetic patients. The diagnosis and treatment of oral lesions will contribute to improve the quality of life of the ESRD-DM patient

1. National Kidney Foundation. K/DOQI clinical practice guidelines for chronic kidney disease: evaluation, classification, and stratification. Am J Kidney Dis 2002; 39: S1–266.
2. Feest TG, Rajamahesh J and Byrne C, Trends in adult renal replacement therapy in the UK: 1982- 2002. QJM 2005; 98: 21-28
3. Löe H, Periodontal disease: the sixth complication of diabetes mellitus. Diabetes Care 1993; 16(1):329-334.
4. Taylor GW and Borgnakke WS, Periodontal disease: associations with diabetes, glycemic control and complications. Oral Dis 2008; 14(3): 191-203.
5. Ship JA., Diabetes and Oral Health: an overview. JADA 2003; 134(suppl):4S-10S.
6. Collins, V. R., Dowse, G. K., Finch, C. F., Zimmet, P. Z. and Linnanae, A. W. Prevalence and risk factors for micro- and macroalbuminuria in diabetic subjects and entire population of Nauru.1989, Diabetes 38, 1602-1610
7. King, H., Aubert, R. and Herman, W., Diabetes mellitus, Diabetes Care 1998; 21, 1414–1431.
8. Mallick NP and Gokal ,. Haemodialysis., Lancet 1999; 353: 737-742
9. Oldenburg B, MacDonald GJ and Perkins RJ, Factors influencing excessive thirst and fluid intake in dialysis patients. Dial Transplant 1988; 17: 21-23

10. Everett KD, Brantley PJ, Sletten C, Jones GN and McKnight GT, The relation of stress and depression to interdialytic weight gain in haemodialysis patients. Behav Med 1995; 21: 25-30
11. Leggat JE, Jr., Orzol SM and Hulbert-Shearon TE , Noncompliance in hemodialysis: predictors and survival analysis. Am J Kidney Dis 1998; 32: 139-145
12. Moran PJ, Christensen AJ and Lawton WJ, Social support and consciousness in hemodialysis adherence. Ann Behav Med 1997; 19: 333-338
13. Fliser D and Haller H, Nephropathie bei Diabetes mellitus Type 2 (Nephropathy in Type 2 DM). Der Internist 2000; 41: 1363 – 73.
14. Stephen Pastan, M.D., and James Bailey, M.D, Dialysis Therapy, N Engl J Med 1998; 338:1428-1437
15. Belmiro Cavalcanti do Egito Vasconcelos , Moacir Novaes , Francisco Aurelio Lucchesi Sandrini , Almir Walter de Albuquerque Maranhão Filho and Leila Santana Coimbra , Prevalence of oral mucosa lesions in diabetic patients: a preliminary study Rev Bras Otorrinolaringol 2008;74(3):423-8.
16. Antunes FS, Graça MA, and Nurkim NL,. Periodontal disease and diabetes mellitus.. Rev Odonto Cienc 2003; 18(40):107-111.
17. Rivera EJ,. Insulin and insulin-like growth factor expression and function deteriorate with progression of Alzheimer's disease:link to brain in acetylcholine. J Alzheimers Dis 2005;8(3):247-268.

18. Paradella TC, Monteiro da Silva CM, and Arisawa EA, O Que o Cirurgião-Dentista Deve Saber Sobre o Paciente Diabético Tipo I,. Rev EAP / APCD 2001;1(3):12-7.
19. Kitabchi AE, Umpierrez GE, Murphy MB, and Kreisberg RA (December 2006)."Hyperglycemic crises in adult patients with diabetes: a consensus statement from the American Diabetes Association". Diabetes Care 29 (12): 2739–48.
20. Burkits, Textbook Of Oral Medicine, 10thedition, 2003, pg 563 – 57521.
21. Scherer LP, and Lobo FMB., Pesquisa do nistagmo / vertigem de posição e avaliação eletronistagmográfica em um grupo de indivíduos portadores de diabetes Mellitus tipo I, Rev Bras Otorrinolaringol 2002;68(3):355-36024.
22. Maia CAS, and Campos CAH, Diabetes Mellitus como causa de perda auditiva., Rev Bras Otorrinolaringol 2005;71(2):208-214.
23. Tommasi AF. Diagnóstico em patologia bucal. 2ª ed. São Paulo, Brasil: Editora Pancast; 1997. p. 527-558.
24. Moore PA, Guggenheimer J, Etzel KR, Weyant RJ, and Trevor O: Type 1 diabetes mellitus, xerostomia, and salivary flow rates. Oral Surg Oral Med Oral Pathol Oral Radiol Endod 2001, 92:281-91.
25. Guggenheimer J, Moore PA, and Rossie K, Insulin-dependent diabetes mellitus and oral soft tissue pathologies, II: prevalence and characteristics of Candida and Candidal lesions. Oral Surg Oral Med Oral Pathol Oral Radiol Endod 2000; 89(5):570-576

26. Sousa RR, Castro RD, and Monteiro CH , O Paciente Odontológico Portador de Diabetes Mellitus: Uma Revisão de Literatura. Pesq Bras Odontoped Clin Integr 2003;3(2):71-77.
27. Yuli M, Muller A, and Yuraima P., Manifestaciones bucales de la Diabetes Mellitus Gestacional- Presentación de dos casos y revision de la literatura. Acta Odontol Venez 2002; 40(2):160-164.
28. Belmiro Cavalcanti do Egito Vasconcelos , Moacir Novaes , Francisco Aurelio Lucchesi Sandrini , Almir Walter de Albuquerque Maranhão Filho ,and Leila Santana Coimbra ,Prevalence of oral mucosa lesions in diabetic patients: a preliminary study Rev Bras Otorrinolaringol 2008;74(3):423-8.
29. Jawa A, Kcomt J, and Fonseca VA. Diabetic nephropathy and retinopathy. Med Clin N Am 2004; 88: 1001 – 1036.
30. Mogensen CE. Progression of nephropathy in long-term diabetics with proteinuria and effect of initial anti-hypertensive treatment. Scand J Clin Lab Invest 1976; 36: 383 – 388.
31. Parving HH, Smidt UM, and Friisberg B, A prospective study of glomerular filtration rate and arterial blood pressure in insulin-dependent diabetics with diabetic nephropathy. Diabetologia 1981; 20: 457 – 461.
32. Cooper ME, Gilbert RE, and Epstein M. Pathophysiology of diabetic nephropathy. Metabolism 1998; 47 (Suppl 1): 3 – 6.
33. Cooper ME. Pathogenesis, prevention, and treatment of diabetic nephropathy. Lancet 1998; 352: 213 – 219.

34. Gonzalez-Albarran O, Gomez, Ruiz E, Vieitez P, and Garcia-Robles R. Role of Systolic blood pressure on the progression of kidney damage in an experimental model of type 2 diabetes mellitus, obesity, and hypertension (Zucker Rats). AJH 2003; 16: 979 – 985.
35. Barnett A. Prevention of loss of renal function over time in patients with diabetic nephropathy. The American Journal of Medicine 2006; 119: 40S – 47S.
36. Kroustrup JP, Gundersen HJ, and Osterby R. Glomerular size and structure in diabetes mellitus. III. Early enlargement of the capillary surface. Diabetologia 1977; 13: 207 – 10.
37. Hostetter TH, Rennke HG, and Brenner BM. The case for intrarenal hypertension in the initiation and progression of diabetic and other glomerulopathies. Am J Med 1982; 72: 375 – 80.
38. Held PJ, The United States Renal Data System's 1991 annual data report: an introduction. Am J Kidney Dis 1991; 18:1-16.
39. Makino H, Phenotypic modulation of the mesangium reflected by contractile proteins in diabetes. Diabetes 1996; 45:488-95.
40. Peter J Watkins, ABC of Diabetes, 5th Edition, 2003, pg no: 65-66.
41. Nielsen S, The clinical course of renal function in NIDDM patients with normal and microalbuminuria. J Intern Med 1997; 241:133-141.
42. Raile K, Diabetic nephropathy in 27,805 children, adolescents, and adults with type 1 diabetes: effect of diabetes duration, HBA1c,

hypertension, dyslipidemia, diabetes onset, and sex. Diabetes Care 2007; 30:2523-2528.

43. Remuzzi G, Clinical practice. Nephropathy in patients with type 2 diabetes. N Engl J Med 2002; 346:1145-1151.
44. Steinke JM, The early natural history of nephropathy in type 1 diabetes: III. Predictors of 5-year urinary albumin excretion rate patterns in initially normo-albuminuric patients. Diabetes 2005; 54:2164-2171.
45. Evans N, and Forsyth E. End-stage renal disease in people with type 2 diabetes: systemic manifestations and exercise implications. Phys Ther. 2004;84:454–463
46. Alba Jover Cerveró, José V. Bagán, Yolanda Jiménez Soriano, and Rafael Poveda Roda, Dental management in renal failure: Patients on dialysis, Med Oral Patol Oral Cir Bucal. 2008 Jul 1;13(7):E419-426.
47. Andrew S. Levey, Juan P. Bosch, and Julia Breyer Lewis, Tom Greene, Nancy Rogers, and David Roth, A More Accurate Method To Estimate Glomerular Filtration Rate from Serum Creatinine: A New Prediction Equation Ann Intern Med. 1999;130:461-470
48. Antonio Nicolucci1, Sheldon Greenfield And Soeren Mattke, Selecting indicators for the quality of diabetes care at the health systems level in OECD countries International Journal for Quality in Health Care; September 2006: pp. 26–30

49. Eleanor Lederer, MD, and Rosemary Ouseph, Chronic Kidney Disease American Journal of Kidney Diseases, Vol 49, No 1 (January), 2007: pp 162-171
50. Chih-Cheng Hsu, Shang-Jyh Hwang, Chi-Pang Wen, , Hsing-Yi Chang, Ted Chen, , Ruei-Shiang Shiu, Shiow-Shiun Horng, Yu-Kang Chang and Wu-Chang Yang, High Prevalence and Low Awareness of CKD in Taiwan: A Study on the Relationship Between Serum Creatinine and Awareness From a Nationally Representative Survey, American Journal of Kidney Diseases Volume 48, Issue 5, November 2006, Pages 727-738
51. Sanjay Kumar Agarwal1, Suresh Chand Dash1, Mohammad Irshad2, Sreebhuasn Raju1, Ravinder Singh1 and Ravinder Mohan Pandey, Prevalence of chronic renal failure in adults in Delhi, India Nephrol Dial Transplant (2005) 20: 1638–1642
52. Jean-Franc¸ and ois Yale, Oral Antihyperglycemic Agents and Renal Disease: New Agents, New Concepts J Am Soc Nephrol 16: S7–S10, 2005.
53. De Rossi SS, and Glick M. Dental considerations for the patient with renal disease receiving hemodialysis. J Am Dent Assoc, 1996; 127:211-219.
54. Jover Cerveró A, Bagán JV, Jiménez Soriano Y, and Poveda Roda R., Dental management in renal failure: patients on dialysis. Med Oral Patol Oral Cir Bucal. 2008;13:419-26.

55. Proctor R, Kumar N, Stein A, Moles D, and Porter S., Oral and dental aspects of chronic renal failure. J Dent Res. 2005;84:199-208.
56. Davidovich E, Davidovits M, Eidelman E, Schwarz Z, and Bimstein E. Pathophysiology, therapy, and oral implications of renal failure in children and adolescents: an update. Pediatr Dent. 2005;27:98-106.
57. Kerr AR, Update on renal disease for the dental practitioner. Oral Surg Oral Med Oral Pathol Oral Radiol Endod. 2001;92:9-16.
58. Sharma DC, and Pradeep AR, End stage renal disease and its dental management. N Y State Dent J. 2007; 73:43-7.
59. Eugene Sobngwi, Sostanie Enoru, Gloria Ashuntantang, Marcel Azabji-Kenfack, Mesmin Dehayem, Arnold Onana, Daniel Biwole, Franc¸ Ois Kaze, Jean-Franc¸ Ois Gautier, and Jean-Claude Mbanya, Day-to-Day Variation of Insulin Requirements of Patients With Type 2 Diabetes and End-Stage Renal Disease Undergoing Maintenance Hemodialysis, Diabetes Care 33:1409–1412, 2010
60. James McKiernan, Rachel Simmons, Jared Katz and Paul Russo, Natural history of chronic renal insufficiency after partial and radical nephrectomy , Urology Volume 59, Issue 6, June 2002, Pages 816-820
61. Burkits, Textbook Of Oral Medicine, 10thedition, 2003, pg 426
62. Miller CS, Little JW, and Falace DA. Supplemental corticosteroids for dental patients with adrenal insufficiency: reconsideration of the problem. J Am Dent Assoc. 2001;132:1570-9.

63. Kasper DL, Braunwald E, Fauci AS, Hauser SL, Longo DL, Jameson JL, Harrison´s Principles of Internal Medicine. New York: McGraw-Hill; 2005.16th edition pg. 1653-63.
64. Robert N. Foley and Allan J. Collins, End-Stage Renal Disease in the United States: An Update from the United States Renal Data System *J Am Soc Nephrol* 18: 2644–2648, 2007
65. Amato D, Alvarez AC, Castañeda LR, Rodríguez E, Avila DM, and Arreola F, Prevalence of chronic kidney disease in an urban Mexican population. Kidney Int 2005;68:S11-7.
66. De La Rosa-García E, Mondragón-Padilla A, Aranda-Romo S, and Bustamante-Ramírez MA. Oral mucosa symptoms, signs and lesions, in end stage renal disease and non-end stage renal disease diabetic patients. Med Oral Patol Oral Cir Bucal 2006;11:E467-73.
67. Chi-yuan Hsu, Eric Vittinghoff, Feng Lin, and Michael G. Shlipak, The Incidence of End-Stage Renal Disease Is Increasing Faster than the Prevalence of Chronic Renal Insufficiency Ann Intern Med. 2004;141:95-101.
68. Emmanuel Villar, Stephen Peter Mcdonald, and Cécile Couchoud, Incidence of Treatment for End-Stage Renal Disease among Individuals with Diabetes in the U.S. Continues to Decline Diabetes Care, Volume 33, Number 5, May 2010 :65-69.

69. Zohreh Hajheydari, and Atieh Makhlough, Cutaneous and Mucosal Manifestations in Patients on Maintenance Hemodialysis A Study of 101 Patients in Sari, Iran, Iranian Journal of Kidney Diseases ,2008;2:86-90.
70. Franois madore, Edmund Lowrie, Carlo Brugnara, Nancy Lew, j. Michael lazarus, Kenneth bridges and William F. Owen, Anemia in Hemodialysis Patients: Variables Affecting this Outcome Predictor, J Am Soc Nephrol 8, 1997:1921-1929.
71. Gall M-A, Hougaard P, and Borch-Johnsen K, Risk factors for development of incipient and overt diabetic nephropathy in patients with noninsulin- dependent diabetes mellitus: prospective observational study. BMJ. 1997;314:783-788.
72. Choy BY, Ho YW, Chau KF, Leung CB, Tsang WK, and Lui SF. Renal replacement therapy for patients with diabetes mellitus in Hong Kong. Hong Kong Journal of Nephrology 2001; 3: 89 – 96.
73. Olugbenga. E. Ayodele , C. Olutayo Alebiosu, and Babatunde. L. Salako, Diabetic Nephropathy A Review of the Natural History, Burden, Risk Factors and Treatment Journal Of The National Medical Association Vol. 96, NO. 11, November 2004 1445-1454.
74. Jorge l. Gross, Mirela j. De azevedo, Sandra p. Silveiro, Lu´is henrique canani, Maria luiza caramori, and Themis zelmanovitz, Diabetic Nephropathy: Diagnosis, Prevention, and Treatment ,Diabetes Care 2005,28:176–188.

75. Thorman R, Neovius M, and Hylander B, Prevalence and early detection of oral fungal infection: a cross-sectional controlled study in a group of Swedish end-stage renal disease patient,. Scand J Urol Nephrol. 2009; 43(4):325-330.
76. Gábor L. Kovács, Diabetic Nephropathy - eJIFCC , 2009,pg-40-52
77. Britt B. Newsome, David G. Warnock, William M. McClellan, Charles A. Herzog, Catarina I. Kiefe, Paul W. Eggers, and Jeroan J. Allison, Long-term Risk of Mortality and End-Stage Renal Disease Among the Elderly After Small Increases in Serum Creatinine Level During Hospitalization for Acute Myocardial Infarction, Arch Intern Med. 2008;168(6):609-616
78. Ritz E, Bergis K, Strojek K, and Keller C., Nephropathy and hypertension in type II diabetes, Med Klin (Munich). 1997 Jul 15;92(7):421-425.
79. Barry I. Freedman, J. Michael Soucie, and William M. Mcclellan, Family History of End-Stage Renal Disease among Incident Dialysis Patients, J Am Soc Nephrol 1997 ,8: 1942-1945,
80. Udayakumar P, Balasubramanian S, Ramalingam KS, Lakshmi C, Srinivas CR, and Mathew AC. Cutaneous manifestations in patients with chronic renal failure (CRF) on hemodialysis. Indian J Dermatol Venereol Leprol 2006;72:119-25
81. Shu-Fen Chuang, Junne-Ming Sung, Shih-Chen Kuo, Jeng-Jong Huang, and Su-Yuan Lee, Oral and dental manifestations in diabetic and

nondiabetic uremic patients receiving hemodialysis, Oral Surg Oral Med Oral Pathol Oral Radiol Endod 2005;99:689-95

82. P. Mosannen Mozaffari, Amirchaghmaghi, and Mortazavi, Oral Manifestations of Renal Patients Before and After Transplantation: A Review of Literature ,DJH 2009; Vol.1, No.1,pg 1-6
83. A M El Nahas, A Masters-Thomas, S A Brady, K Farrington, V Wilkinson, Z Varghese, and J F Moorhead, Selective effect of low protein diets in chronic renal diseases British Medical Journal Volume 289 17 November 1984, pg 1337-1341
84. Agarwal SK, Dash SC, and Irshad M, Prevalence of chronic renal failure in adults in Delhi, India. Nephrol Dial Transplant.2005;20:1638–1642.
85. Robert N. Foley and Allan J. Collins, End-Stage Renal Disease in the United States: An Update from the United States Renal Data System , J Am Soc Nephrol 18: 2644–2648, 2007
86. Sowell SB, Dental care for patients with renal failure and renal transplants. JADA 1982;104:171-7.
87. Carl W, and Wood RH. The dental patient with chronic renal failure. Quintessence Int1976;7:9-15.
88. Hovinga J, Roodvoets AP, and Gaillard J. Some findings in patients with uremic stomatitis. J Maxillofac Surg 1975; 3:125-7.
89. Vesterinen M, Ruokonen H, Furuholm J, Honkanen E, and Meurman JH., Clinical questionnaire study of oral health care and symptoms in

diabetic vs. non-diabetic predialysis chronic kidney disease patients. Clin Oral Investig. 2011 Apr 1:453-45994

90. Gavaldá C, Bagán JV, Scully C, Silvestre FJ, Milián MA, and Jiménez, Renal hemodialysis patients: oral, salivary, dental and periodontal findings in 105 adult cases. Oral Dis 1999;5:299-302
91. De La Rosa-García E, Mondragón-Padilla A, Irigoyen-Camacho ME, and Bustamante-Ramírez MA. Oral lesions in a group of kidney transplant patients. Med Oral Patol Oral Cir Bucal 2005;10:196-204.
92. Safia A. Al-Attas, and Soliman O. Amro, Candidal colonization, strain diversity, and antifungal susceptibility among adult diabetic patients, Ann Saudi Med 2010; 30(2): 101-108
93. E. de la Rosa García, S. Cruz Mérida and A. Mondragón Padilla, Tooth loss in diabetic patients with and without end-stage renal disease and dialysis Nefrología 2008; 28 (6) 645-648
94. Abe M, Okada K and Soma M., Antidiabetic agents in patients with chronic kidney disease and end-stage renal disease on dialysis: metabolism and clinical practice, Curr Drug Metab. 2011 Jan; 12(1):57
95. Jean-Francoise Yale, Oral Antihyperglycemic Agents and Renal Disease: New Agents, New Concepts J Am Soc Nephrol 2005,16: S7–S10,
96. M. Greenwood and J. G. Meechan, General medicine and surgery for dental practitioners Part 6: The endocrine system British Dental Journal 2003; 195: pg 129–133

97. Janelle C Nisbet, Joanna M Sturtevant, and Johannes B Prins, Metformin and serious adverse effects, MJA Vol 180 19 January 2004, Pg- 53-54

98. Devasmita Choudhury and Cynthia Luna-Salazar, Preventive health care in chronic kidney disease and end-stage renal disease, nature clinical practice april 2008 vol no 4, pg 194-206

99. Farman A.G, Central papillary atrophy of the tongue; Oral Surg. 1977: 43: Pg 48-58.

100. Arendorf TM and DM Walker: Oral Candidal population in Health and disease. Br Dent J, 1979: 147: 267-272.

101. Jay D. Shulman, and Francisco Rivera-Hidalgo, The prevalence of oral mucosal lesions in U.S. adults J Am Dent Assoc, 2004 Vol 135, No 9, 1279-1286.

102. Prabhu SR, Wilson DF, Daftary DK and Johnson NW. Oral diseases in tropics. 1st edition, 1993 25-125.

103. Ronald D. Perrone, Nicolaos E. Madias, and Andrew S. Levey, Serum Creatinine as an Index of Renal Function: New Insights into Old Concepts, CLIN.CHEM.38/10, 1933-1953 (1992)

104. Jean-Luc Orsonneau, Catherine Massoubre, Marie Cabanes, and Patrick Lustenberger, Simple and Sensitive Determination of Urea in Serum and Urine, CLIN.CHEM.38/5,619-623(1992)

105. Anjan basak, Development Of A Rapid And Inexpensive Plasma Glucose Estimation By Two-Point Kinetic Method Based On Glucose

Oxidase-Peroxidase Enzymes, Indian Journal Of Clinical Biochemistry, 2007 / 22 (1) 156-160

106. Hans Berninger and Roger Smith, A Modification of the Spectrophotometric Determination of Carbon Monoxide in Blood Enabling Direct Determination of Per Cent Hemoglobin Saturation, Clinical Chemistry Vol. 5, No. 2, 1959, 127-134.

107. Borch-Johnsen K, Andersen PK, Deckert T. The effect of proteinuria on relative mortality in type 1 (insulin-dependent) diabetes mellitus. *Diabetologia* 1985;28(8):590-6.

108. Groop PH, Thomas MC, Moran JL, Wadén J, Thorn LM, Mäkinen VP, et al. The presence and severity of chronic kidney disease predicts all-cause mortality in type 1 diabetes. *Diabetes* 2009; Jul; 58(7):1651-8.

Master chart of ESRD -DM Patients

						DIABETES MELLITUS				MEDICATION			INVESTIGATION				ORAL LESIONS PRESENT IN ESRD															
S.NO	AGE	SEX	TOBACCO HABITS	DIET	HYPERTENSION	IDDM	NIDDM	DURATION	FAMILYHISTORY	METFORMIN	SULPHONYLUREA	INSULIN	SERUMCREATININE	FASTING BLOOD SUGAR LEVEL	BLOOD UREA LEVEL	HB LEVEL	SABURRAL TONGUE	FISSURED TONGUE	CANDIDIASIS	DRY AND FISSURED LIPS	PETECHIAE/ECCHYMOSES	SMOOTH TONGUE	BURNINGTONGUE	UREMIC STOMATITIS	HERPES SIMPLEX	APHTHOUS	ANGULARCHELITIS	UREMIC FETOR	XEROSTOMIA	PALE MUCOSA	STOMATIS MEDICAMENTOSA	LICHENPLANUS	UNPLEASANT TASTE
1	35	M	S	R		1		10			1		2.4	239	112	15																1
2	59	M	T	NV			1	20		1			8	190	59	6.4	1															1
3	27	F		NV			1	12			1		12	126	124	4.8							1		1			1	1	1			1
4	25	F		NV		1		10			1		4.8	220	81	6.2							1						1				1
5	49	F		R	1		1	20			1		6.3	99	90	4.6			1									1	1	1			
6	58	M	T	NV			1	20			1		2.6	148	42	5.4																
7	55	M	T	NV	1		1	15		1			7.1	120	110	9.8												1	1	1			
8	60	F		NV			1	15	1		1	1	2.8	144	47	7.7												1	1	1			
9	31	F		NV		1		17			1		2.2	170	43	8.8					1							1		1			1
10	61	F		NV			1	20		1			3	132	45	7.4	1				1								1	1			1
11	38	M	S	NV			1	10		1			2.3	154	48	5.7					1							1	1	1			1
12	45	F		V	1		1	20			1		12	160	180	7.3												1	1	1			1
13	47	M	S	NV	1		1	2		1			12	96	122	9.9										1		1	1	1	1		1
14	39	M	S	NV			1	6		1			5.8	75	93	11	1											1	1				1

15	55	M	S	R	1		1	30		1			11	142	155	16	1						1				1	1		1		
16	46	M	S	NV			1	20		1			2.5	206	45	8.8						1								1	1		
17	18	M		NV	1	1		15			1		6.7	197	87	8.5				1										1			
18	46	F		V	1		1	10	1	1			4.4	185	96	8																
19	50	M	S	NV	1		1	30	1	1			11	270	162	7.2	1		1									1	1	1			
20	45	F		NV	1		1	30	1	1			5.6	210	110	8.8			1			1								1			1
21	46	F		NV	1	1		30			1		8.2	240	74	7.8				1			1				1	1	1	1			1
22	55	F		NV			1	10		1			7.4	180	52	8.9														1		1	
23	43	M	t	NV	1		1	10		1			15	96	150	4.9		1		1			1							1			1
24	68	F		NV	1		1	2			1		3	111	67	8.9				1							1	1	1				1
25	72	F	t	NV			1	10		1			2.8	170	122	13											1						
26	30	F	t	NV			1	16			1		2.8	84	123	8									1		1				1	1	
27	65	M	s	V			1	15	1	1			2.9	190	54	13				1		1					1	1					
28	48	F		V			1	15	1	1			2.4	71	76	13				1	1						1						1
29	61	F	t	V			1	10		1			4.8	117	49	7.9				1							1						1
30	55	F		V			1	15	1		1		4	202	50	9.9			1														1
31	56	M	s	V			1	19	1	1			3.1	125	60	8.9	1			1							1	1					
32	47	M	s	V			1	5	1	1			3	210	65	10																	
33	60	M	s	NV			1	25	1	1			3.4	125	66	7.6	1					1	1				1	1					
34	60	M		V			1	8		1			4.8	190	111	11											1						
35	47	F		NV			1	20				1	2.4	337	40	11																	
36	65	M	s	V			1	20	1	1			3.6	142	60	13											1						
37	58	F		NV			1	20			1		3.4	110	45	12												1	1				
38	60	F	t	V	1		1	11	1	1			2.5	137	111	11											1						
39	30	M		NV		1		19	1		1		4.2	203	123	12	1		1														
40	66	F	t	NV			1	10		1			5.2	120	40	9.7	1										1						
41	58	F	t	NV			1	30		1			4.9	184	48	10	1								1				1				
42	70	M	t	V	1		1	20				1	3.2	186	48	11					1												
43	60	F		NV			1	2		1			5.2	140	40	12						1					1	1					
44	45	M	t	V			1	4		1			3.3	188	77	14				1									1				
45	60	F	t	V			1	40			1		3.3	198	142	16					1								1				1
46	85	F		V	1		1	2		1			3.7	93	42	12			1	1							1		1				

47	50	M		V			1	9		1			3.7	164	48	7.9			1									1	1				1
48	42	M	s	NV			1	5		1			4.5	223	65	8.2			1											1			1
49	36	F		NV		1		26			1		2.2	82	111	10			1			1								1			1
50	55	F		NV	1		1	10	1		1		2.8	300	76	12	1	1	1	1	1							1		1			1
51	38	M		NV			1	8		1			3.1	214	48	9.4			1										1	1			1
52	60	F		NV			1	10			1		2.3	86	40	7			1			1								1			
53	53	F		NV			1	5	1	1			2.8	200	77	8.5	1											1	1	1			
54	60	M	s	NV			1	30	1	1			3.1	210	40	12	1		1		1									1			1
55	55	F		NV			1	5				1	4	91	76	12			1									1		1			
56	40	M		NV			1	25	1	1			2.2	160	76	11												1				1	
57	55	F		NV			1	2			1		4	242	40	11	1											1	1				1
58	43	F		NV			1	3		1			2.4	210	35	12												1	1				
59	60	M	s	NV	1		1	40			1		2.8	80	76	13	1											1					1
60	48	F		V			1	5			1		4.7	210	115	13				1	1		1					1	1			1	
61	46	F		NV			1	5			1		3.1	93	132	13				1								1					
62	47	f		NV		1		22			1		4	221	123	11	1						1					1	1				
63	17	F		NV		1		10			1		2.7	108	69	12																	
64	38	F		NV		1		10				1	3.9	152	133	12					1	1											1
65	28	M	s	V			1	12			1		4	183	76	8												1					1
66	51	M	s	NV			1	10			1		4.2	84	67	9.8	1						1		1				1				1
67	41	M	t	NV	1	1		20	1		1		2.1	134	43	9.8			1		1		1						1				1
68	65	F	t	NV	1		1	15		1			4.4	294	52	9.9	1		1									1	1				1
69	59	M	s	NV			1	15			1		4.7	101	67	7.9			1		1						1	1					
70	39	F		NV		1		35	1		1		2.6	157	56	12				1	1							1	1	1			1
71	21	F		V		1		15			1		2.3	204	55	9			1											1			1
72	61	F		V			1	48		1			11	92	123	8.6						1									1	1	
73	47	M	t	V			1	22	1	1			13	116	132	7.6							1					1		1	1		1
74	53	F		NV			1	8	1		1		4.7	239	98	8.8														1			1
75	65	F	t	NV	1		1	10		1			4.2	108	40	13				1	1									1			
76	81	M		NV	1		1	5		1			2.4	265	44	13			1		1									1			1
77	79	M	s	NV			1	4		1			3.8	125	69	13						1										1	
78	71	F		NV	1		1	21		1			4.2	86	47	8.5														1			

79	62	F		V			1	8		1			2.9	215	44	14				1												
80	52	F		V			1	15			1		4.5	243	55	11												1		1		
81	36	M		V		1		25			1		2.5	150	53	13														1		1	
82	37	M		NV			1	11		1			3	82	66	16					1				1			1		1		
83	61	M	s	NV			1	8			1		3.5	151	40	12			1									1	1			
84	64	F		V			1	31			1		2.9	119	45	13														1		
85	59	F		V	1		1	6	1		1		3.9	135	42	10												1	1	1		
86	50	F		NV			1	8		1			4.8	300	45	11				1									1			
87	54	F		NV		1		20				1	2.8	140	66	6.8					1							1				
88	47	F		NV			1	7		1			2.8	250	48	8.8				1									1	1		1
89	41	F		NV			1	14	1		1		2.2	98	40	8.4			1													1
90	60	M	s	NV			1	3			1		11	130	154	5.5				1												1
91	55	F		NV			1	6			1		2.3	175	66	7.3				1							1		1			1
92	60	F		NV			1	10		1			2.3	240	42	5.6			1	1								1	1	1			1
93	60	F	t	NV	1		1	25				1	2.6	158	45	10						1	1							1			1
94	42	F		V			1	7		1			2.9	128	41	7												1	1				1
95	46	F		NV			1	8			1		2.3	93	68	6			1										1	1			
96	72	M	s	NV	1		1	11		1			2.5	120	67	7.3	1				1		1					1	1				
97	40	F		V			1	5		1			2.1	220	60	6																	
98	52	F		V			1	6		1			2.2	130	40	9.4							1										
99	38	M		NV	1	1		25	1			1	3.2	80	43	8												1	1	1			
100	26	M		NV		1		12				1	2.2	140	67	8.8				1									1				1

Master chart of NESRD -DM Patients

						DIABETES MELLITUS				MEDICATION			INVESTIGATION				ORAL LESIONS PRESENT IN NESRD																
S.NO	AGE	SEX	TOBACCO HABITS	DIET	HYPERTENSION	IDDM	NIDDM	DURATION	FAMILYHISTORY	METFORMIN	SULPHONYLUREA	INSULIN	SERUMCREATININE	FASTING BLOOD SUGAR LEVEL	BLOOD UREA LEVEL	HB LEVEL	SABURRAL TONGUE	FISSURED TONGUE	CANDIDIASIS	DRY AND FISSURED LIPS	PETECHIAE/ECCHYMOSES	SMOOTH TONGUE	BURNINGTONGUE	UREMIC STOMATITIS	HERPES SIMPLEX	APHTHOUS	ANGULARCHELITIS	UREMIC FETOR	XEROSTOMIA	PALE MUCOSA	STOMATIS MEDICAMENTOSA	LICHENPLANUS	UNPLEASANT TASTE
1	30	M		NV		1		10					1	117	46	15													1				
2	21	F		NV		1		15				1	0.8	106	22	11						1											
3	20	M		NV		1		20				1	1	93	28	14													1	1			
4	48	M		NV			1	2					1.2	120	32	15	1		1														1
5	35	F		V			1	4						110	38	13				1			1								1	1
6	44	F		V			1	10					0.9	105	28	12	1		1										1			
7	32	M		NV		1		14				1	1	185	31	15										1						
8	23	F		V		1		13				1	0.9	190	30	9				1												
9	32	M		NV			1	1	1				1.1	289	40	10			1														1
10	68	M		V			1	4	1			1	1	260	30	11	1		1	1												
11	32	F		V		1		5				1	0.7	146	26	11			1	1		1	1						1			
12	70	M	s	V			1	26				1	0.7	130	36	12							1						1			
13	56	M		V			1	2		1			1.8	202	30	13				1												

14	36	F		V	1		1	3					0.8	108	25	11			1													
15	55	F		NV			1	2					1.1	226	30	11				1										1	1		
16	44	F		NV			1	2					1	70	30	12																
17	38	F		V			1	2					1.4	160	26	13														1	1		1
18	73	M	s	NV			1	15					0.8	90	29	12														1	1		
19	43	F		NV			1	5					0.7	180	28	12			1											1			1
20	65	F		NV	1		1	4				1	1.2	170	30	13				1													
21	37	M		NV		1		25				1	0.7	185	21	12									1								1
22	18	M		NV			1	1					1.2	88	30	11										1							
23	20	M		V		1		2				1	0.7	213	27	8			1											1			
24	54	M		V			1	1	1				0.9	192	30	10																1	
25	50	M		V			1	2					1	150	35	11																	1
26	46	F		V			1	4					1.2	152	45	10				1													
27	42	F		V	1		1	6					0.9	102	29	11																	1
28	23	M		V		1		11	1			1	1.1	82	35	12			1											1			1
29	70	M	s	V			1	1					1.4	261	15	15	1													1			1
30	15	F		NV		1		7	1			1	0.8	270	34	16						1	1										
31	49	F		V			1	12					1.1	161	24	16			1	1													1
32	52	M	s	NV			1	1					1.1	268	28	14				1										1	1		
33	34	F		NV			1	1					1	210	32	16				1							1						1
34	60	M	s	NV			1	5				1	1.2	156	46	13							1										
35	36	M		V		1		25				1	1.2	131	38	13																	
36	28	F		V			1	1					0.9	82	25	13			1												1		
37	46	F		V	1		1	2	1				0.9	220	28	13							1							1			
38	31	F		V		1		14	1			1	0.7	175	22	15							1							1			
39	28	F		V		1		10				1	0.8	100	23	12				1			1				1			1			
40	40	F		V			1	3					0.8	170	28	11			1											1			1
41	52	M	s	V			1	2					1.1	293	30	11				1			1				1						
42	36	F		V			1	3				1	1.8	229	40	11														1			1
43	27	F		V		1		16		1		1	0.6	132	25	13														1			
44	24	M		V		1		15					1	208	22	11			1											1			
45	45	F		V			1	2	1				1	110	30	13														1			1

46	46	F		V			1	1	1				0.9	200	28	13					1								1				1
47	51	F		V			1	7					0.7	130	25	13													1				1
48	60	F		V	1		1	12					1	116	34	13			1														
49	45	F		V			1						0.6	170	22	11				1							1		1				
50	15	F		V		1		5				1	0.8	105	28	10			1	1		1						1	1	1			
51	27	F		V			1	1					0.8	98	24	10													1				
52	45	F		V			1	1					0.7	110	27	11									1				1				
53	22	F		V			1	1				1	0.9	104	28	14																	1
54	50	F		V			1	1					0.9	90	28	15			1										1				
55	35	F		V			1	1					0.9	130	38	16				1							1		1				
56	40	M	s	V		1		40				1	0.6	108	21	13																	1
57	22	F		V		1		2				1	0.8	210	29	12					1								1				1
58	55	F		V	1		1	1					0.7	160	23	11			1														1
59	42	F		NV	1		1	4					0.9	102	29	13				1							1			1			1
60	82	F		NV	1		1	1					0.9	103	30	12				1							1		1				1
61	51	F		V			1	4					0.7	318	18	11			1	1											1	1
62	25	F		V		1		1				1	1	89	31	11			1														
63	45	M		V	1		1	4	1			1	0.7	180	25	11			1			1											
64	38	F		V			1	3	1				1	200	35	13			1										1				
65	56	F		V			1	3	1				0.7	154	25	15				1		1					1						
66	57	F		V	1		1	6					1	104	25	13										1							
67	45	F		V			1	1					1	90	30	9			1										1				
68	59	F		V			1	9				1	1.1	148	27	11																	1
69	32	F		V			1	1					0.9	90	22	10				1													
70	45	M	s	V			1	2				1	1.1	178	36	12			1														
71	55	M	s	V			1	1				1	1.1	92	31	13																	1
72	58	F		V			1	6				1	0.9	94	35	12																	1
73	27	F		NV			1	3					0.9	74	22	10			1										1				
74	45	F		V	1		1	6					0.9	96	30	13		1															1
75	54	F		V	1		1	2					1	95	21	13			1										1				1
76	21	M		V		1		12	1			1	0.7	65	24	14													1	1			
77	26	M		V		1		12				1	1	83	32	12													1				1

78	19	F		V		1		5				1	1.2	330	32	14													1				
79	16	M		V			1	2				1	0.7	150	26	13																	1
80	15	M		V			1	4				1	0.9	236	30	15									1								1
81	60	F	t	V			1	3					0.8	161	20	12						1											1
82	46	F	t	NV			1	1	1	1			0.6	187	23	11														1			1
83	34	M		V			1	1					1	134	28	11			1										1				
84	66	M	s	V			1	6					0.9	83	30	11										1							
85	44	F	t	V			1	3	1				0.9	210	29	11																	1
86	52	M	t	V			1	8	1				1	135	30	12			1				1						1			1	1
87	52	F	t	V			1	3				1	1	120	24	13																	
88	35	F		V			1	2					1	173	33	12																	1
89	24	F		V			1	1					1	78	21	11																	
90	39	M	s	V			1	1				1	1.1	104	38	15													1				1
91	36	F		V			1	5					1.2	145	21	14			1														
92	26	M	s	V	1		1	9				1	0.8	305	16	15																	1
93	27	M		NV			1	4				1	0.9	280	30	13													1				1
94	56	F	t	NV			1	3	1				0.7	123	24	12	1													1			
95	33	F		V			1	2	1	1			0.7	103	25	12				1													
96	30	F		V	1		1	5		1			0.9	212	29	12											1		1				
97	50	M		V			1	2	1			1	1.2	220	34	12																	
98	28	M		V			1	16	1			1	0.8	141	26	12																	
99	20	F		V			1	4	1	1			1.1	73	28	13																	
100	70	M	s	V			1	5	1			1	0.9	132	29	11		1															

RAGAS DENTAL COLLEGE & HOSPITAL

2/102, East Coast Road, Uthandi, Chennai - 600119

DEPARTMENT OF ORAL MEDICINE & RADIOLOGY

CASE SHEET PROFORMA

Date:

Name:

Age:

Sex:

Occupation:

Income:

Address:

Phone number:

Tobacco related habits:

Dietary habits:

Medical history:

1. Systemic disease:
2. Diabetes mellitus:

a) Duration of diabetes:

b) Family history:

c) Medication:

Investigations:

Serum creatinine level:

Blood glucose level: fasting sugar:

Serum urea level:

Oral lesion present:

- Saburral tongue :
- Candidiasis:
- Dry and fissured lips:
- Petechiae or Ecchymoses:
- Smooth tongue:
- Burning tongue:
- Ulcerative or uremic stomatitis:

- Herpes simplex:
- Angular cheilitis:
- Uremic fetor:
- Xerostomia:
- Pale mucosa:
- Other lesions:

CONSENT LETTER

I ……, the undersigned hereby given my consent for the performance of diagnostic test on myself to evaluate the incidence of oral mucosal lesions in the diabetic patients with renal disease conducted by Dr.B.Senthil under the able guidance of Dr.(Capt.).S.Elangovan ,M.D.S., Professor, Department Of Oral Medicine And Radiology, Ragas Dental College And Hospital,Chennai-600119. I have been informed and explained the status of my disorder ,evaluation procedure, risk involved and likelihood of success .I also understand and accept this as a part of the study protocol there by voluntarily unconditionally freely give my consent without any fear or pressure in mentally sound and conscious state to participate in the study

Witness/representative Patient signature

ஒப்புதல் படிவம்

---------- என்கின்ற நான், சென்னை, ராகாஸ் பல் மருத்துவக் கல்லூரி மற்றும் மருத்துவமனையின் வாய் மருத்துவம் மற்றும் ஊடுகதிர் துறையின் பேராசிரியர் மரு.கேப்டன், எஸ்.இளங்கோவன் அவர்களின் மேற்பார்வையில், முதுநிலை(M.D.S) பட்டப்படிப்பு பயிலும் மரு.பி.செந்தில் அவர்கள் மேற்கொள்ளும், ''நீரிழிவு நோயாளிகளின் சிறுநீரக கோளாறால் ஏற்படும் வாய் சம்மந்தமான நோய்களை கண்டறிதல்''என்கின்ற ஆராய்ச்சிக்கான பரிசோதனைகளுக்கு என்னை உட்படுத்துவதற்கு எனது மனமுவந்த பரிபூரண சம்மத்தினை அளிக்கிறேன். மேலும் எனக்கு என்னுடைய நோயின் தன்மையைப் பற்றியும், அதனால் ஏற்படக் கூடிய விளைவுகளைப் பற்றியும் எடுத்துக் கூறப்பட்டுள்ளது எனவும், இந்த பரிசோதனைக்கு நான் எந்தவித அச்சமுமின்றி தன்னிச்சையாகவும், தெளிவான முழு மனதுடன் என்னுடைய பரிபூரண சம்மதத்தினை அளிக்கிறேன் என இதன் மூலம் தெரியப்படுத்துகிறேன்.

சாட்சியாளர்கள் இப்படிக்கு

தேதி